NEW SERIES, VOL. XXXIX MARCH, 1938 No. 3

The American Journal of Surgery

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EDITORIAL

DANGEROUS LEGISLATION

One of the most spectacular and most needed reforms in medicine in recent years has been the country-wide campaign against syphilis. As a result of the publicity successfully given a hitherto unmentionable topic, the so-called "social diseases" have been dragged out into the full glare of the light. "Syphilis" is no longer a word taboo in most newspaper and magazine offices (although the radio still maintains a few qualms). High school and college students of both sexes have learned so much about the cause and menace of syphilis that the subject has become a commonplace, no longer something to be discussed in whispers in a dark corner. The people of the land have learned that syphilis is not uncommon; that mothers about to bring babies into the world may have it, and that the newly born child, as a result, may be syphilitic; that all this is unnecessary because it can be prevented; and finally, that if the disease actually exists, it can be cured in the great majority of cases.

For a long time physicians in many prenatal clinics have examined the blood of pregnant women routinely for evidence of syphilis. Until recently, however, this has not been true in private practice. Now, as a result of the publicity given the subject, many physicians do make a habit of such routine blood examinations, and we learn, it is not unusual these days for a pregnant woman to request such a test of her own accord. All in all, splendid work has been done and much good has resulted. Within the past year a greater advance has been made in educating the laity concerning this menace to health than had been achieved during the past two thousand years.

This educational campaign, so long delayed and so fruitful of results, should undoubtedly be continued without let-up. It should be intelligently directed to enlighten and educate.

But it ought not be so conducted that it produces "syphilis scares" or creates "syphilophobias." Certain types of humans are too ready to become "syphilophobiacs"; traces of this disorder are already discernible as a result of the activities of the over-imaginative press.

As with everything that is good, well meaning, but often short-sighted and half-informed, individuals seize upon the idea, go off at a tangent, and rush to design laws which, by fiat, will create a diseaseless and perfect humanity. Many of the proposed laws are idiotic, silly, and loaded with dynamite. In their fervor, the proposers feel that mankind must be saved at all cost and that this desirable end may be achieved by compulsion and laws.

In many States, legislation has been proposed with the aim of compelling all those contemplating marriage, as well as all pregnant women, to undergo blood tests. The clinical judgment of the physician seems no longer to be sufficient to determine the necessity of such a test; instead, an invariable rule is set up and the test is done, willy-nilly.

This brings up several important considerations: Who will be responsible (should such laws be passed) for the diagnosis? Will it be the laboratory worker who examined the blood? Suppose a report from a laboratory with a reading of one, two, or four plus breaks up a projected marriage, but later it is discovered that the report was in error and the examined person did not have syphilis—is it the technician who must defend the malpractice suit which may result? If not he, then who is responsible? How many blood tests are to be demanded of each individual prior to a diagnosis? Does a single negative test rule out syphilis? Are all antigens of the same sharpness? One can go on for a long time in this vein. Indeed, would a law be constitutional, if it compelled a citizen, even against his will, to have a needle pushed into one of his veins and blood withdrawn?

It might be well for the strong men in medicine to reason these points out with legislators who propose measures along these lines, in the hope that the bills will die before birth. Otherwise, the good accomplished by the syphilis educational program may have been in vain. Cranks and charlatans will undoubtedly find the opportunity to shout their claims to high heaven and exploit and confuse the public by means of irrelevant side-issues.

Our task is to educate the public along sane, conservative, scientific lines, and not allow the real issue to be befogged. Also, let us educate physicians to recognize the clinical signs of syphilis, to be familiar with the indications for laboratory testing, and to know how properly to treat the disease when diagnosed.

But the silly debutante who bursts into print to urge her parents, her old maiden aunts and all within reach of her advice to have their blood tested should be firmly led away and rendered into a state of innocuous desuetude. And, to the same limbo we might relegate the reckless lawmaker who would save humanity at all costs and with complete publicity, by the use of laws of compulsion.

T. S. W.

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ORIGINAL ARTICLES

HUMAN CRANIOVERTEBRAL DYNAMICS*

JULIUS LOMAN, M.D.

Junior Visiting Neurologist, Beth Israel Hospital; Instructor in Neurology, Tufts College Medical School; Research Associate, Boston State Hospital

BOSTON, MASSACHUSETTS

IN a consideration of the dynamic phenomena occurring within the craniovertebral cavity, the clinician is concerned mainly with that fluid peculiar to the cavity, namely, the cerebrospinal fluid. He is not so directly aware of those other two more active components, the arterial and venous pressures, yet it is upon these latter forces that the dynamic state of the cerebrospinal fluid largely depends.

It has been only recently that the cerebral vascular components have been experimentally approachable in the human being. The introduction by Myerson and his associates[1] of the techniques of puncture of the internal jugular vein and the carotid artery have made it possible to study not only the metabolism of the brain but also pressure reactions of the cerebral venous and arterial components.[2,3] Furthermore, by combining the jugular puncture technique with Gibb's thermoelectric determination of blood flow,[4] a great deal of data have been gathered concerning the cerebral circulation of man under various conditions.

The concepts of the craniovertebral dynamics resulting from the use of these methods, together with those obtained by the classical punctures of the subarachnoid space, form the main basis of the present communication. It has been pointed out in a previous paper[5] that lack of agreement between the results of the dynamic studies on the human and animal craniovertebral cavity can often be explained by: (1) differences in vascular responses in the two species, for example, the assumption of the upright position and the need for facile and frequent changes in posture by man calls into play a more complex dynamic mechanism than necessary in animals; (2) differences in experimental conditions, for example, the usual necessity for preliminary narcosis, and the more technical or operative interference in animals may make evaluation of the subsequent results difficult or impossible. These latter objections have been eliminated or reduced to a minimum in the results referred to in the present communication.

The Three Fluid Systems of the Craniovertebral Cavity and Their Environment. The cerebrospinal fluid has no active circulation in the same sense as the vascular system. Being very slowly produced at the choroid plexus, either as a dialysate or as a true secretion, it enters the ventricular system and flows toward the subarachnoid space, where its pressure is slightly lower than at its source, but slightly higher than the pressure in the venous system into which it empties.[2,6,7] Whether the cerebrospinal fluid finally escapes into the blood stream by way of the arachnoid villi, the cortical veins, the perineural lymph channels, or by way of more than one of these is still a much debated question. Once the fluid comes into contact with the arteries

* From the Division of Psychiatric Research, Boston State Hospital, Abraham Myerson, M.D., director; aided by grants from the Commonwealth of Massachusetts and the Rockefeller Foundation.

and veins of the subarachnoid space, it now very sensitively reflects the pressure changes occurring within these vessels.

Because of their enclosure within a bony cavity, the vascular and cerebrospinal fluid systems appear at first glance to be subject to the peculiar physics of a closed box. When one examines the system more closely, however, one has definitely to modify such a rigid conception. A brief review of the internal and external environment of the craniovertebral cavity will make this more apparent. One may speak of the craniovertebral contents as having two types of elements: first, the static or inelastic; and second, the dynamic or elastic. In the first category are included the brain, the cerebrospinal fluid, and the meninges; in the second category, the blood vessels, particularly the thin walled veins. Enclosing these elements are the cranial and vertebral bony walls having many openings for the passage of the arteries and the veins. Thus, penetrating the cranial cavity, are the carotid and vertebral arteries and their branches, the internal jugular veins, and the emissary veins. Along the spine are foramina for the passage of veins and arteries which connect the intradural with the extravertebral vessels. Thus, dynamic phenomena occurring on one side of the bony barrier are readily reflected to the other side of the barrier. By means of these vascular communications the craniovertebral contents are subject to atmospheric changes, particularly through the medium of the thin walled cranial and spinal veins. Some examples of the mechanism by which extracraniovertebral phenomena by way of the vascular system affect the dynamics of the cavity are the following:

The Direct Relationship between the Venous and Cerebrospinal Fluid Pressures.

1. Changes in intrathoracic pressure. Inspiration causes a decrease in intrathoracic pressure which is reflected in a lowered intracranial venous pressure; expiration results in the opposite phenomena. Coughing and straining likewise increase the intrathoracic and intracranial venous pressures. Reflections of these changes may be observed by noting the pressure alterations in a manometer attached to a needle which is in the internal jugular vein.[2] These changes in venous pressure are directly transmitted to the cerebrospinal fluid pressure.

2. Changes in intra-abdominal pressure. Coughing, straining, and manual abdominal pressure increase the intra-abdominal venous pressure, a change which is reflected not only to the intraspinal veins, but also to the cerebral veins, hence the increase in cerebrospinal fluid pressure.[2]

3. Compression of the jugular veins. When either the internal or external jugular vein is compressed, the damming back of the blood into the cranial cavity causes an immediate rise in cerebral venous pressure which is immediately reflected to the cerebrospinal fluid pressure. This direct and sensitive relationship may be demonstrated by the combined measurement of the spinal fluid and internal jugular venous pressures.[2] The Queckenstedt phenomenon would be impossible were it not for the presence of the elastic components of the craniovertebral cavity, that is, the thin walled, compressible and distensible cerebrospinal veins. When the venous blood is dammed back by jugular compression into the cranial cavity, the consequent dilatation of the cerebral veins causes a compensatory displacement of fluid from the ventricles and craniosubarachnoid spaces into the spinal subarachnoid space. This latter space is able to accommodate the expelled cerebrospinal fluid because of the presence of the many compressible spinal veins. There is a limit, however, to which the cerebrospinal fluid pressure can rise once the maximum distensibility of the veins is reached and once the other venous pathways increase in their capacity to take up the excess return blood flow. The effects of prolonged compression of the cerebral venous pathways will be discussed later.

A phenomenon similar to the Queckenstedt occurs when abdominal compression

is employed. Here a damming back of venous blood into the spinal veins by way of the abdominal venous connections creates a dilatation of the former vessels and a consequent rise in spinal fluid pressure. Since the capacity of the craniovertebral cavity is fixed because of its bony covering, dilatation of the spinal veins must be compensated for by constriction of the veins elsewhere in the cavity. The cerebrospinal fluid pressure, however, is

Table I

Relationship of cerebrospinal fluid and internal jugular pressure (in mm. of water) in the horizontal position*

Case	C.F.P.	I.J.P.	Difference
1	80	70	10
2	140	120	20
3	100	70	30
4	150	140	10
5	90	80	10
6	170	90	80
7	150	135	15
8	110	90	20
9	110	90	20
10	140	30	110
11	180	115	65
12	140	80	60
13	140	75	65
14	100	90	10
15	155	70	85
16	100	80	20
17	170	70	100
18	110	80	30
19	85	40	45
20	190	75	115
21	85	40	45
22	60	0	60
23	160	90	70
24	150	40	110
25	110	70	40
26	70	40	30

* C.F.P. indicates cerebrospinal fluid pressure; I.J.P., internal jugular pressure.

increased equally throughout the subarachnoid space. The reflection craniad of this increased spinal venous and cerebrospinal fluid pressures is, of course, impaired or prevented in the presence of a spinal cord block.

4. A bodily change illustrating the exquisite and direct relationship between the venous and cerebrospinal fluid pressure is postural alterations. That the cerebrospinal fluid pressure changes *pari passu* with the changes occurring in the venous pressure may be illustrated by the following experiments. If one simultaneously measures the internal jugular and cisternal pressures of a subject in the recumbent position, it will be observed that the venous pressure is somewhat lower than the spinal fluid pressure. (Table I.) Similar observations in animals have been made by Weed and Hughson,[6] Bedford,[7] and others, who utilized the sagittal sinus as an index of the cerebral venous pressure. Upright tilting now results in a marked fall in cisternal and internal jugular pressures to atmospheric. Tilting the body to the vertical head-down position results in a marked rise in both pressures to 400–500 mm. of water. When the body is tilted from the lateral recumbent to the supine

Table II

Comparison of the lumbar pressures (in mm. of water) in the hands-and-knees ("quadruped") and lateral recumbent positions

Case	Hands-and-Knees Pressure	Lateral Pressure
1	−20	75
2	95	160
3	15	95
4	30	145
5	45	160
6	10	100
7	20	140
8	20	105
9	75	140
10	85	105
11	−15	90
12	20	120
13	105	185
14	75	160
15	30	75
Average.....	40	124

position, both pressures are definitely increased. A shift in posture from the lateral recumbent to the hands-and-knees or "quadruped" (head and spine in horizontal alignment) produces a marked fall in internal jugular venous and cerebrospinal fluid pressures to atmospheric

pressure.[5] That these variations in the cerebrospinal fluid pressure depend upon changes in venous pressure will be made clearer later. (Tables II, III, and IV.)

Let us first consider the factors which influence the venous pressure in relationship to the craniovertebral cavity when the position of the body is altered:

TABLE III
CISTERNAL PRESSURE (IN MM. OF WATER) SHOWING THE VARIATIONS WITH POSTURAL CHANGE

Case	Supine Pressure	Vertical Head-up Pressure	Vertical Head-down Pressure	Lateral Pressure	Sitting Pressure
1	160	−10	495		
2	140	20	515		
3	150	−40	550	135	−25
4	145	5	545	140	30
5	120	−15	555	55	−55
6	120	−45	550	75	−45
7	145	25	435	120	10
8	120	−15	425	75	−50
Average..	137	− 9	509	100	−22

1. The position of the right side of the heart with respect to the neural axis. In the supine position, the heart, which may be considered the venous outlet, and the great veins of the chest and abdomen lie above the subarachnoid space, and in the reverse position ("quadruped") these vessels lie below the subarachnoid space. As a result, the intradural venous pressure will be relatively higher in the former than in the latter position.

TABLE IV
COMPARISON OF THE CISTERNAL AND INTERNAL JUGULAR VENOUS PRESSURES (IN MM. OF WATER) IN THE HORIZONTAL AND "QUADRUPED" POSITIONS

	Case No.	35	39	44	45	48
Horizontal	Cisternal	120	145	75	175	145
	Jugular	105	65	60	90	80
Quadruped	Cisternal	50	60	30	50	5
	Jugular	15	10	20	55	50

2. The volume of blood in the great veins. In the supine position the volume of blood in the abdominal and thoracic veins is increased. This tends to increase their pressure, which is reflected back to the intradural veins. The opposite changes occur in the "quadruped" position.

3. The weight of the viscera lying on the great veins. The weight of the intestinal tract, the liver, etc., lying against the great veins tend to compress them, to impede the flow of blood, and to raise the venous pressure in their tributaries. These factors would operate in opposite directions in the supine and "quadruped" positions.

4. The tension of the thoracic and abdominal muscles. The venous pressure would alter in opposite directions during inspiration and expiration and during contraction and relaxation of the abdominal wall. As a result of these factors, the cerebrospinal fluid pressure is directly influenced.

Although these factors readily explain the variations that the cerebrospinal fluid pressure undergoes in the horizontal positions, it is more difficult to explain the changes in pressures which occur in the vertical positions, for here there must be considered the hydrostatic effect of the superimposed column of spinal fluid itself. In the literature, there is a wide variation of normal pressure readings given for the spinal fluid pressure in the sitting position. For example, Barré and Schrapf[8] give a range of lumbar pressure in this position from 150 to 400 mm. of water; Zilberlast-Zand[9] gives a figure of 200 to 300 mm.; Pappenheim,[10] a figure between 200 and 300 mm. higher than that in the horizontal position. Ayer[11] and Fremont-Smith and Kubie[12] state that the lumbar pressure is double when a subject changes from the lateral to the sitting position. A customary statement found in the literature is that the lumbar pressure taken in the sitting position is dependent upon the height of the column of spinal fluid above the site of

puncture. The studies of Loman, Myerson and Goldman,[5] reported in 1935, and more recently those of von Storch, Carmichael and Banks,[13] indicate a close relationship between the lumbar sitting pressure and the lumbar cisternal distance. In animals, on the other hand, Weed[14] found that one-third to one-fourth of the superimposed column of spinal fluid is active in increasing the lumbar sitting pressure. It is obvious that the hydrodynamics mechanism involved in man and in animals is quantitatively different. The hydrodynamic mechanism involved in the upright position cannot be reduced to any simple pressure distance relationship, but it is apparent that it must involve an individual variable factor or factors. This factor, most of the evidence indicates, is the change in intradural venous pressure in relationship to the site of puncture. Von Storch, Carmichael and Banks[13] come to an identical conclusion after repeating experiments similar to those of Loman, Myerson and Goldman.[5]

Thus, it would be more nearly correct to state that the spinal fluid pressure obtaining in the upright position is not directly related to the height of the column of superimposed fluid, but rather to the pressure reflected to that fluid by the hydrostatic and elastic changes occurring in the venous wall surrounding the fluid. This conclusion, based on indirect observations, cannot, of course, be proved without measurements of the pressure changes in the intradural spinal veins during postural alterations, a procedure which appears to be impossible at the present time. To what degree the various factors which affect the intradural venous pressure operate to affect the cerebrospinal fluid pressure in the upright position, it is again impossible to state. Such a conception as advanced, however, appears best to explain the cerebrospinal fluid pressure in the upright position and probably accounts for the wide variations in pressure readings given by various observers.

Although most experimental work demonstrates the great dependence of the cerebrospinal fluid pressure upon the venous pressure, there is a definite difference in opinion regarding the reverse relationship. Weed and Hughson,[6] for example, state that variations in the pressure of the cerebrospinal fluid pressure affect, within physiological limits, the pressure within the cerebral veins. Becht,[15] on the other hand, found in a large series of experiments on animals that no such relationship existed. It must be stressed that the conclusions regarding the effect of the changes in spinal fluid pressure upon the venous pressure have been largely based on the results of the effects of hypertonic and hypotonic solutions, which introduces other complicating factors, particularly the disturbance in water balance in the nervous system.

Furthermore, it must be remarked that variations in the torcular or sagittal venous pressure do not necessarily correspond to those occurring in the thin cerebral veins, since the former vessels are rigidly enclosed in contrast to the latter. We have observed in several cases, for example, that variations in cerebrospinal fluid pressure have no effect on the pressure in the internal jugular vein punctured close to the jugular bulb, a point comparable to the venous sinus. That the above criticism holds is borne out by the experiments of Gibbs,[16] who utilized the orbital venous pressure as an index of cerebral venous pressure. Measurements of the pressure in the orbital veins are most analagous to those taken in the thin walled cerebral veins, for both sets of vessels are similarly enclosed in a bony vault. Gibbs clearly demonstrated that the orbital venous pressure closely and directly follows changes in cerebrospinal fluid pressure.

The Indirect Relationship between the Arterial and Cerebrospinal Fluid Pressures. Most investigators agree that there is a less direct relationship between cerebrospinal fluid and arterial pressure than between the former and venous pressure

(Fremont-Smith and Kubie,[17] Weed and Hughson,[6] Loman and Myerson,[3,5] and many others). As many workers have demonstrated, it is only when sudden and marked changes in arterial pressure occur that the cerebrospinal fluid pressure becomes altered. The change in cerebrospinal fluid pressure under such conditions is obviously due to two factors secondary to the change in arterial pressure: (1) the marked change in caliber of the cerebral vessels; and (2) the change in venous pressure. On the other hand, a steady high arterial pressure, as pointed out by Fremont-Smith and Merritt,[18] does not affect the cerebrospinal fluid pressure, unless there is associated a high venous pressure or other complicating pathologic factors.

Since it is necessary that alterations of sudden and great magnitude occur in the arterial pressure before the cerebrospinal fluid pressure becomes altered, conversely it is difficult to conceive how changes in the latter pressure can alter the arterial pressure. There is no definite proof that such a phenomenon takes place. In the writer's experience, drainage of large amounts of cerebrospinal fluid has no effect upon the carotid pressure and therefore probably not on the cerebral arterial pressure. From the clinical point of view, there appears to be no basis for the often expressed fear that therapeutic lumbar puncture following cerebral hemorrhage increases the bleeding in the affected artery. Certainly the reduction of cerebrospinal fluid pressure, for example, of 50 mm. of water, can have little effect on an artery carrying a pressure of approximately 1000 to 1500 mm. of water.

We have used the following hydrodynamic schema to explain the effect of alterations of the venous and arterial pressures upon the cerebrospinal fluid pressure. The cerebrospinal fluid pressure may be considered to be surrounded by four walls: (1) the dura; (2) the arteries; (3) the capillaries; and (4) the veins. Of these the dura is the most rigid and can have little to do with affecting the cerebrospinal fluid pressure. The arteries, having a relatively high pressure, also form a relatively rigid wall. The capillaries form a wall of less rigidity, and the veins a wall which is exceedingly elastic. Under ordinary physiologic conditions, such as during changes in posture, the arterial pressure can affect the cerebrospinal fluid pressure but little, because whatever slight bulging of the arterial wall occurs will be readily compensated for by a corresponding and partial collapse of the venous wall. Thus, the arterial pressure will be fully supported by the arterial wall and the change in pressure will not be transmitted to the cerebrospinal fluid. These same factors will operate to prevent a decrease in arterial pressure from being transmitted to the cerebrospinal fluid pressure. Nor will the capillary pressure influence the cerebrospinal fluid pressure except as it changes the balance between fluid production and absorption. Furthermore, simple changes in the volume of the capillary bed will be readily compensated for by changes in volume of the venous bed. Any increase in venous pressure, on the other hand, will cause a bulging of the venous wall. Neither capillary, arterial, nor dural wall will support the venous pressure. The change in venous pressure, therefore, will be transmitted directly to the cerebrospinal fluid pressure.

Influence of Drugs on the Cerebrospinal Fluid and Intracranial Vascular Pressures. If this simple hydrodynamic concept is kept in mind, it is not usually difficult to explain the variations in cerebrospinal fluid pressure following the administration of drugs, although here other factors influencing its pressure, such as changes in osmotic pressure or in fluid production, may also operate. The baffling question of whether the cerebrospinal fluid pressure is elaborated by a process of secretion or by dialysis has failed to be answered by drug experimentation, as emphasized by Becht several years ago. He concluded from his work that increases in cerebrospinal fluid pressure following administration of the drugs used is adequately explained on a hydrodynamic basis, that is, as a result of

changes in either venous or arterial pressure. Unless all the various factors which may modify the cerebrospinal fluid pressure are controlled, it is impossible to state definitely the cause of the changes in that pressure. Some of the drugs which we have carefully studied and which have at times been considered by former investigators to cause changes in actual secretion of cerebrospinal fluid will now be briefly considered.

1. Ether. An understanding of the cerebrospinal fluid responses following the use of this anesthetic is important, since it has been commonly used in animal experimentation preliminary to dynamic studies. Ether has been shown by Forbes, Wolff and Cobb,[19] Myerson and Loman,[2] and others to produce a profound change in the cerebral vascular and cerebrospinal fluid pressures. Among other changes, it produces a marked congestion of the brain. For this reason, ether interferes with the interpretation of the pure hydrodynamic effect of the drug subsequently studied. Thus, Myerson and Loman[2] have shown that anesthetization with ether creates a great simultaneous and equivalent rise in both the jugular and cerebrospinal fluid pressures, associated with a fall in carotid arterial pressure.

Narcosis by sodium amytal[20] seems to be far superior to ether in the study of craniovertebral dynamics since it affects the cerebrospinal fluid and venous pressures but little, although it does cause a temporary fall in arterial pressure.

2. Adrenalin. Intravenous injections of this drug cause a rapid and marked rise in cerebrospinal fluid, internal jugular venous and arterial pressures.[3] Forbes and Wolff[21] have demonstrated that associated with the great rise in arterial pressure, there is a coincident cerebral vasodilatation when the drug is given intravenously. That the cerebrospinal fluid and venous pressures are very closely related is indicated by the fact that they are practically identical in the extent of their rise. On the other hand, when the pial vessels are irrigated with adrenalin, there is vasoconstriction.

3. Pituitrin. Pure extract of the posterior pituitary body influences the hemodynamics of the craniovertebral cavity in a manner similar to adrenalin, although with much less catastrophic effects. Wolff[22] has noted a rise in cerebrospinal fluid and venous pressures as well as a slight cerebral vasoconstricting action of the drug in cats. That the increase in cerebrospinal fluid pressure is readily explained on a rise in cerebral venous pressure is demonstrated by the experiments of Loman and Myerson,[3] in which intravenous injections of pituitrin caused simultaneous increases in jugular and cerebrospinal fluid pressures, even though the arterial pressure fell, showing the direct relationship between these two pressures and the independence of the cerebrospinal fluid pressure and the arterial pressure. In Wolff's experiments on cats, during the time of the cerebral vasoconstricting effect of the drug, there was noted a temporary fall in cerebrospinal fluid pressure followed by a rise, while the arterial pressure was maintained.

4. Caffeine. In unanesthetized human beings, caffeine regularly causes a fall in cerebrospinal fluid pressure. Little alteration, however, occurs in either the arterial or the jugular venous pressures.[3] The cause of the fall in cerebrospinal fluid pressure is apparently explained on the basis of cerebral vasoconstriction, as noted by Gibbs, Gibbs and Lennox[23] in animals narcotized by sodium amytal. Finesinger,[24] however, found that in animals narcotized by sodium amytal, injections of caffeine caused a cerebral vasodilatation accompanied by an increase in cerebrospinal fluid pressure. On the other hand, on postetherized animals a vasoconstriction, accompanied by a fall in cerebrospinal fluid pressure, was observed by the above author. The latter's experiments demonstrate the variable results of the drug according to the anesthetic utilized.

5. Histamine. The effects of this drug on the cerebral vessels, depending upon the

anesthetic used, is well demonstrated by the experiments of Lee,[25] Forbes, Wolff and Cobb,[19] Loman and Myerson.[3] Lee, who

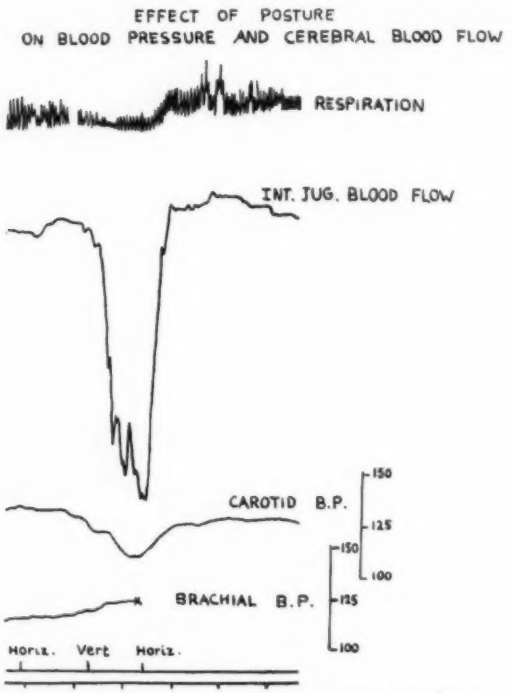

CHART 1. Tilting from the horizontal to the upright position: Note the direct relationship between the carotid (direct) pressure and the blood flow through the internal jugular vein. The brachial arterial (direct) pressure rises while the carotid pressure falls.

anesthetized his animals with ether, observed a cerebral vasoconstriction associated with a fall in cerebrospinal fluid pressure following injections of histamine. Under similar conditions, Forbes, Wolff and Cobb noted the same phenomena as Lee. But when the former workers used sodium amytal as an anesthetic, histamine injections caused a cerebral vasodilatation, associated with a rise in cerebrospinal fluid pressure. In unanesthetized individuals, Loman and Myerson noted regularly a rise in cerebrospinal fluid pressure accompanied by a rise in internal jugular venous pressure.

6. Amyl Nitrite. This vasodilator, like histamine, effects a fall in arterial pressure at a time when there is a cerebral vasodilatation and a rise in cerebrospinal fluid pressure. In man, inhalations of the drug result in a rise in internal jugular venous and cerebrospinal fluid pressures, while the arterial pressure falls.[3] Histamine and amyl nitrite are thus excellent examples of the divorce in relationship between the arterial and the cerebrospinal fluid pressures, the latter following either the venous pressure or the cerebral vasodilatation.[27]

7. Oxygen and Carbon Dioxide. The work of Wolff and Lennox[28] stresses the importance of carbon dioxide as a cerebral vasodilator par excellence. As these authors point out, inhalations of carbon dioxide cause a cerebral vasodilatation and result in an increase of speed and volume in blood passing through the brain. Consequently, the venous blood becomes more arterial-like, permitting a diminution of respiratory effort, a fall in blood pressure, and a decrease in caliber of the cerebral vessels. Thus, as Wolff and Lennox state, on a constant though abnormal gas mixture, an animal is able to adjust to the new conditions with a return of the cerebral circulation to its normal condition. Coincident with the great vasodilatation caused by a rich carbon dioxide mixture, there is, of course, a marked increase in cerebrospinal fluid pressure. On the other hand, oxygen acts very inertly on the cerebral circulation, showing slight vasoconstriction accompanied by a slight fall in cerebrospinal fluid pressure.

Influence of Variations in Osmotic Forces and in the Production-Absorption Mechanism on the Cerebrospinal Fluid Pressure. Thus far, we have considered two of the three factors upon which physiologic variations in cerebrospinal fluid pressure depend, namely, changes in the venous pressure and changes in caliber of the cerebral vessels. The third factor consists of changes in osmotic pressure. Under ordinary physiologic conditions, the osmotic equilibrium between blood and cerebrospinal fluid is so difficult to upset and changes so slowly that it can have very little to do with variations in cerebrospinal fluid pressure. The passage of fluid through the capillary membrane continues at a very slow and definite rate, depending upon the salts in

the blood and the cerebrospinal fluid and the pressure gradient between the cerebrospinal fluid and the blood.

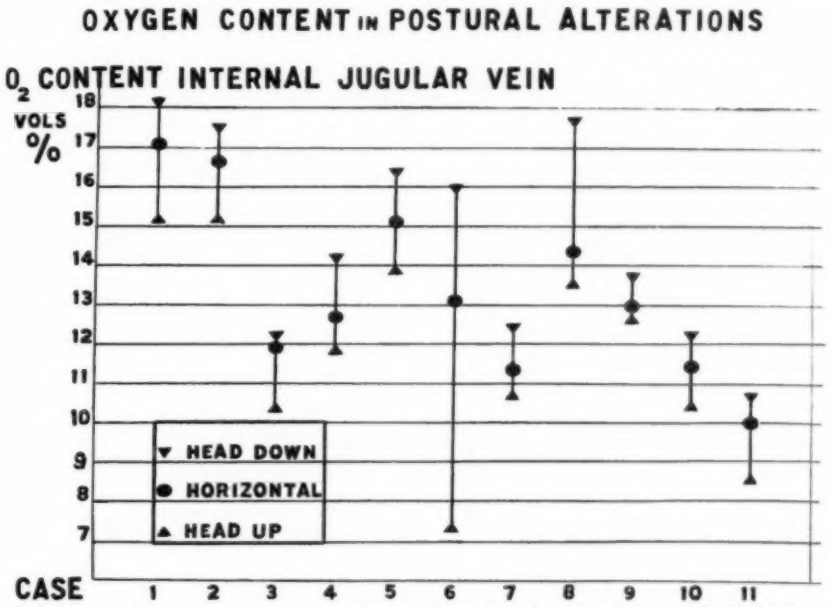

Chart 11. The oxygen content in the internal jugular blood is regularly higher (less uptake of oxygen by the brain associated with increased cerebral flow) in the head-down position than in the head-up position (greater uptake of oxygen associated with a slower cerebral flow). Case 6 shows the lowest oxygen content in the upright position, at which time syncope occurred.

Experimentally, one may upset the fluid balance, as is well known, by the administration of hypertonic or hypotonic solutions. Thus, as first demonstrated by Weed and McKibben[29] and corroborated by many others, the injection of hypertonic solutions of salt or glucose withdraws fluid not only from the subarachnoid spaces, but also from the parenchyma of the brain itself. On the other hand, the introduction of hypotonic solutions into the circulation creates the opposite phenomenon, that is, the passage of water from the blood into the central nervous system, so that the cerebrospinal fluid pressure becomes increased.

In those pathologic conditions accompanied by an increase in intracranial pressure, the fall in cerebrospinal fluid pressure created by injections of most hypertonic solutions is unfortunately followed by a secondary wave of increased pressure, often even higher than the original. In the author's experience, as well as that of others, however, the normal cerebrospinal fluid pressure is diminished with great difficulty by the use of glucose. The least objectionable of the hypertonic solutions has been found by Gregersen and Wright,[30] Masserman,[31,32] and others, to be sucrose. It has been shown by these workers that the greater efficiency of this sugar in decreasing the cerebrospinal fluid pressure is due to the fact that sucrose, unlike glucose, does not pass over into the cerebrospinal fluid, so that the latter fluid does not become hypertonic in relationship to the blood, thus preventing a secondary rise in cerebrospinal fluid pressure. The osmotic balance between the blood and the cerebrospinal fluid may be disturbed under conditions of altered salt, sugar, and water metabolism, such as may be seen in diabetes, states of dehydration, and during some severe infections. Again, in experimental hyperinsulinism, when the blood becomes hypotonic, the cerebrospinal fluid pressure becomes elevated to a great height. Associated with the fall in blood sugar, there occurs a relative cerebral anoxemia as indicated by a decreased

uptake of oxygen by the brain (Dameshek and Myerson[33]).

Under physiologic conditions it is difficult or impossible to maintain an elevated cerebrospinal fluid pressure by directly changing the fluid volume, such as by injecting a small amount of isotonic salt solution or cerebrospinal fluid into the subarachnoid space. There is, within a few minutes following the injection, a rapid resumption of the original cerebrospinal fluid pressure. Such an increase in volume is quickly taken care of by a readjustment of the production-absorption mechanism. Once the cerebrospinal fluid pressure remains elevated well above the pressure in the cerebrospinal veins, there is obviously interference in the drainage mechanism. Unless, however, there is a widespread interference with the absorption mechanism, as is possible from a thick exudate in meningitis, there may be sufficient venous bed to take care of the cerebrospinal fluid absorption. Even prolonged complete obstruction of the external jugular veins or lateral sinuses may not cause a breakdown of the elaboration-absorption mechanism, at least for some hours, as is exemplified by the very instructive experiments of Bedford.[8] This author demonstrated that the complete obstruction of both external jugular veins or both lateral sinuses in cats resulted, first, in a great rise in both the torcular and cerebrospinal fluid pressures; and second, in a gradual fall to normal of the cerebrospinal fluid pressure. The torcular venous pressure remained several times its original height, although now much lower than its original increase following the establishment of a collateral venous circulation. Such experiments indicate that a normal cerebrospinal fluid pressure is possible in the presence of a high intracranial venous pressure, when the latter pressure has been sufficiently prolonged to provoke changes in the production-absorption mechanism.

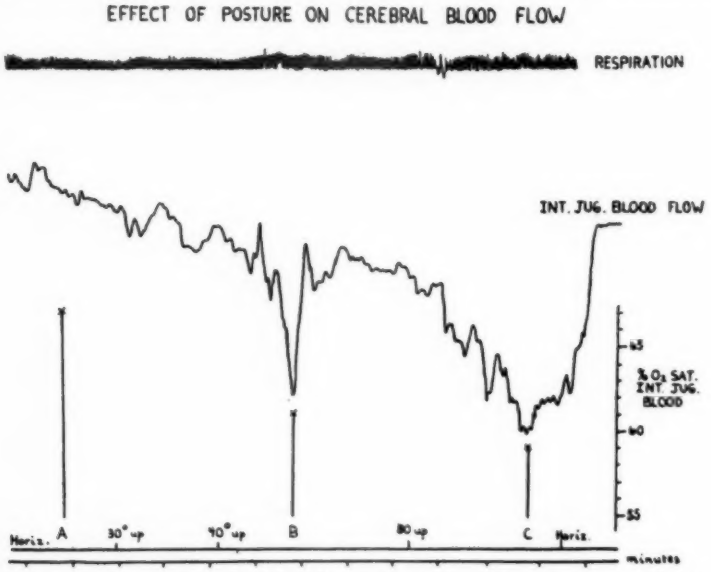

Chart III. With a gradual fall in cerebral blood flow during upright tilting, there is associated a diminution of oxygen saturation in the internal jugular blood. Samples of blood taken at A, B and C in the horizontal, 30 degrees and 80 degrees head-up positions respectively.

Involved in this unusual relationship between the cerebral venous and cerebrospinal fluid pressures, is the fact that despite a high venous pressure, which by itself would tend to increase the production of cerebrospinal fluid because of a reflected increase in capillary pressure, and despite the high intracranial venous pressure which per se would tend to diminish the absorp-

tion of cerebrospinal fluid, there is, nevertheless, a fall to normal of the cerebrospinal fluid pressure. It is difficult to explain, under the above experimental conditions, the fall in cerebrospinal fluid pressure on any other basis than a diminution in volume of cerebrospinal fluid consequent on an increased rate of absorption of fluid, although, as pointed out by Bedford, it is hazardous without more direct data to make definite statements in regard to intracranial volume and pressure relationships. It may be that under such circumstances, an accelerated absorption of cerebrospinal fluid occurs in the spinal veins in which the pressure is much lower than in the cranial veins. At any rate, the ability of the cerebrospinal fluid to return to normal in the face of a prolonged venous obstruction is a remarkable example of fluid homeostasis. A more generalized obstruction of the venous pathways, such as is possible in widespread meningitis, may, of course, cause a cessation of flow of cerebrospinal fluid if the pressure in the subarachnoid space becomes equal to the ventricular pressure. In the presence of an incomplete venous obstruction, however, the flow from the ventricles to the subarachnoid space must continue, as pointed out by Fremont-Smith,[34] since the pressure in the ventricles remains at a higher level than in the subarachnoid space. This increase in filtration pressure is possible, since the capillary pressure becomes increased from the reflection of the increase in intracranial venous pressure. The flow thus can be maintained in spite of the increased cerebrospinal fluid pressure.

Diminution in cerebrospinal fluid pressure, such as by direct withdrawal of cerebrospinal fluid, results not only in a fall of cerebrospinal fluid pressure, but also in a drop in the cerebral venous pressure, since these two pressures are closely related. As the fluid is withdrawn, the cerebrospinal veins are observed to dilate,[35] tending thus to maintain the original intracranial volume without, however, a return of cerebrospinal fluid pressure to the original level. Such a lack of direct relationship between the total craniovertebral volume and intracranial pressure is again seen in

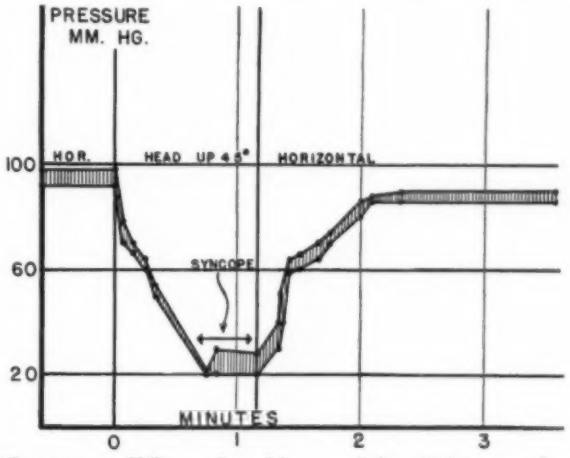

Chart iv. Effect of sudden upright tilting on the carotid pressure: The maneuver was unexpected by the subject who went into syncope at the time the pressure was slightly above 20 mm. Hg.

the experiment of subarachnoid injection of a few c.c. of isotonic salt solution. Although here there must necessarily be a compensatory "collapse" of the most elastic tissue (that is, the veins) to accommodate the injected fluid with a resumption of the original volume, the cerebrospinal fluid pressure rises sharply until the fluid balance is resumed. Although it takes but a few minutes for the original intracranial pressure to be reestablished following injection of isotonic solution into the subarachnoid space, it takes a much longer time for the resumption of pressure to occur following the withdrawal of a similar amount of fluid. The former represents more of an emergency mechanism than the latter. Rapid removal of greater amounts of fluid may cause not only cerebral congestion, but also actual edema of the brain (Masserman[36]). Yet these changes in volume are not associated with a reciprocal response in the cerebrospinal fluid pressure.

Factors Influencing Cerebral Blood Flow. Of more direct importance than simple variations in cerebrospinal fluid pressure are alterations in the cerebral blood flow.*

*These experiments were done in collaboration with Dr. F. A. Gibbs.

Thus, it is important to know not alone how high or how low is the level of the cerebrospinal fluid pressure, but what

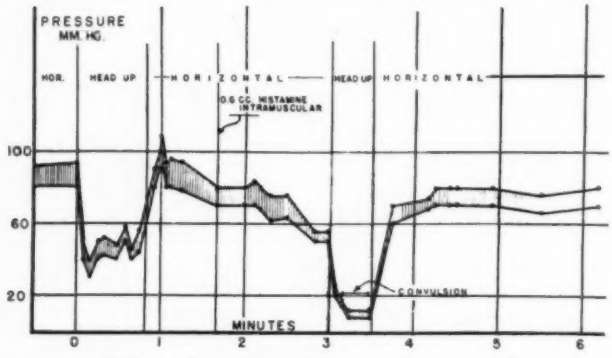

Chart v. Effect of upright tilting on the carotid pressure in a subject who previously received an injection of histamine. Clonic movements of the body occurred when the carotid pressure was 12 mm. Hg.

occurs under such circumstances to the circulation of the blood through the brain. The most direct factor in the control of the cerebral blood flow is the height of the systemic arterial pressure. Other factors being equal, the greater the height of the arterial pressure, the greater the cerebral blood flow. The nicety of this relationship is well brought out in postural experiments in which the carotid and brachial arterial pressures are measured simultaneously with the cerebral blood flow.[37] (Chart I.) Great variations in arterial pressure may, however, be accompanied by variations in the diameter of the cerebral vessels, a factor which in itself greatly influences the cerebral blood flow. Thus, intravenous injections of epinephrine not only cause a great rise in systemic arterial pressure but the rise, if great enough, will overcome the cerebral vasoconstriction with a resulting greater increase in the cerebral flow. In some circumstances, in fact, the cerebral flow may be speeded up by cerebral vasodilatation even though the general arterial pressure falls, provided the fall is not too great. Such a reaction may be produced by intravenous injections of histamine or inhalations of amyl nitrite, drugs which affect the vessels of the brain very sensitively.[23]

A cerebral vasodilator *par excellence* is carbon dioxide, which causes tremendous increase in cerebral blood flow with but little associated change in systemic arterial pressure. The nicety of the relationship between the cerebral blood flow and the arterial pressure is demonstrated during postural alterations by means of the technique of the direct carotid measurements and the thermoelectric determination method of Gibbs, in which the internal jugular puncture is utilized. When a subject is passively tilted from the horizontal to the upright position, there is observed a fall in carotid pressure varying usually from 20 to 40 mm. of mercury and *pari passu* a fall in cerebral blood flow.[37,38] Opposite and comparable alterations in pressure and flow occur during head-down tilting.[38] Still greater changes in pressure and flow are prevented from occurring, probably mainly through the activity of the splanchnic vasomotor mechanism. The fall in blood flow through the brain, which attends upright tilting, is associated with a greater "uptake" in oxygen through that organ, as manifested by a decrease in oxygen saturation in the internal jugular blood.[37] (Charts II and III.) The opposite phenomena are associated with head-down tilting. It is probable that during such changes in position a slower blood flow or a more rapid blood flow through the brain is compensated for by an increase or diminution, respectively, in oxygen utilization by the brain.

Under unusual circumstances the assumption of the upright position may result in such a great fall in cerebral arterial pressure with a corresponding deficit of oxygen utilization by the brain that either fainting or convulsions may ensue. Thus, fainting occurred in one of the author's subjects, who was suddenly and unexpectedly tilted from the horizontal to the upright position. Here the fear and the circulatory disadvantage of the brain in the upright position created a marked slowing in cerebral blood flow attendant upon the marked drop in cerebral arterial pressure (to 20 mm. of mercury). (Chart IV.) The same burden may be thrown upon

the cerebral circulation when vasodilator drugs, such as histamine or sodium nitrite, are administered and upright tilting is performed. (Chart v.) It is remarkable, however, that unconsciousness or alarming symptoms do not appear until the carotid pressure falls to a level of about 20 mm. of mercury. Comparable to this low pressure is the low oxygen saturation of the internal jugular blood (11 to 25 volumes per cent) seen under the above circumstances.[37,39,40]

The defective vasomotor mechanism in arteriosclerosis may also be demonstrated by measuring the carotid pressure during upright tilting of the body. Two abnormal tipping reactions in this condition have been observed (Loman, Myerson, Dameshek and Goldman[39]): (1) an unusual fall in carotid pressure without the usual normal and immediate partial return in pressure towards its horizontal level while the upright position is maintained; and (2) an increasing fall in carotid pressure on repeating the tilting. These reactions indicate the poor tone of the vascular system in arteriosclerosis, the burden being most sensitively borne by the cerebral circulation. (Chart VI.) Such experiments graphically explain the symptoms of dizziness, fainting, and (rarely) convulsions, brought on by sudden changes in the position of the head in arteriosclerosis. In many cases of post-traumatic neurosis or vasomotor neurosis, dizziness or fainting under the same conditions is common, probably on a similar basis of a defective vasomotor system. Thus, in such conditions upright tilting, if unaccompanied by good vasoconstriction of the splanchnic area, may result in an abnormal fall in cerebral arterial pressure and a fall in cerebral blood flow ending in a relative cerebral anemia.

Such knowledge of postural reactions may be applied in the treatment of cerebral vascular accidents. Thus, in cerebral hemorrhage due to hypertension, it would, from a theoretical point of view, seem best to place the patient with the head at a higher level than the rest of the body, since the cerebral arterial pressures is in this position at a lower level than it is in the horizontal position. On the other hand, in

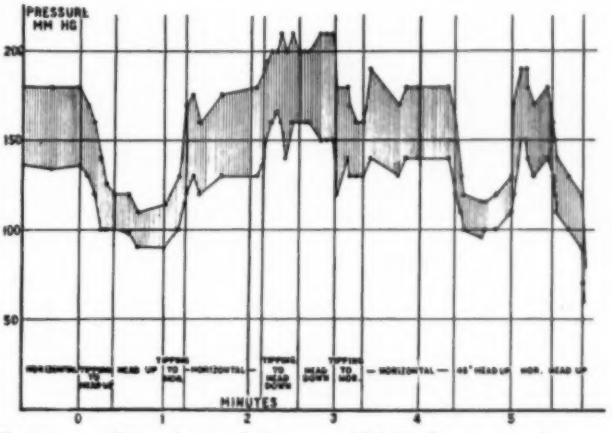

Chart VI. Reactions of the carotid blood pressure in an arteriosclerotic: Note the wide variations in pressure, particularly from the horizontal to the upright position. Finally, on repetition of head-up tilting, the pressure fell to 60 mm. Hg. at which time syncope was imminent.

cerebral thrombosis, since the aim is to increase the collateral circulation to a part suddenly deprived of its main blood supply, it would appear best to place the patient with the head at a lower level than the rest of the body, a position in which the cerebral arterial pressure and blood flow are at higher levels than in the horizontal position.

How important a rôle the cerebral vasomotor system plays in adjusting the cerebral circulation during postural alterations, is questionable. Although there is now no doubt, as a result of the histologic demonstrations of Hassin,[41] and Penfield,[42] and Stöhr,[43] and the physiological experiments of Wolff, Forbes, Cobb, and others,[44] of the existence of such a system of nerves, their importance in the regulation of the cerebral circulation should not be exaggerated. Such a system of nerves may be considered to have a fine adjustment function, in contrast to the splanchnic vasomotor system which, though coarser in its regulatory mechanism, plays the major rôle in maintaining the cerebral circulation.

Two factors have been mentioned which are important in the control of the blood

flow through the brain, namely, the height of the systemic arterial pressure and the width of the cerebral vascular bed. A third factor which may alter the cerebral flow is the pathologic level of the intracranial pressure, such as occurs in the presence of brain tumor or following severe head injury. Gradual or moderate increases in intracranial pressure may not interfere with the cerebral flow so long as the arterial pressure remains sufficiently high. We have observed, for example, that gradually increasing the cisternal pressure to a level of 400 mm. of water had no effect on the flow through the internal jugular vein. (Chart VII.) During such moderate increases in the height of the cerebrospinal fluid pressure, it is probable, as pointed out by Wolff and Blumgart,[45] that a compensatory cerebral vasodilatation is sufficient to maintain an adequate cerebral blood flow. Even when the intracranial pressure is slowly raised to a point close to the systemic arterial level, the local circulation gradually adjusts itself (Wolff and Forbes,[46] Wolff and Blumgart[45]). It is only when the brain is suddenly overwhelmed by great increases in intracranial pressure that the systemic arterial pressure responds to aid the flow of cerebral blood.

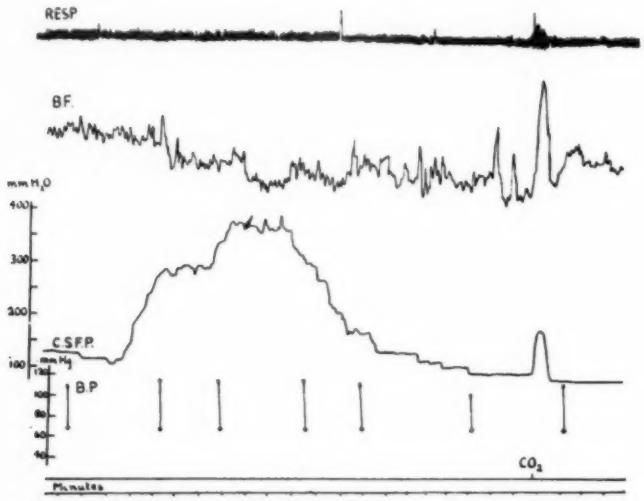

CHART VII. The effect of increasing the cerebrospinal fluid pressure (C.S.F.P.) to approximately 400 mm. H_2O on the blood flow (B.F.) through the internal jugular vein: No definite decrease of blood flow is noted. The inhalation of carbon dioxide causes a sharp rise in blood flow associated with an increase in cerebrospinal fluid pressure.

SUMMARY

The human craniovertebral cavity, although practically completely surrounded by bone, is subject to relatively wide variations in pressure. The activity of the cerebrospinal fluid is dependent upon: (1) a stable production-absorption mechanism; (2) the phenomena occurring in the intracraniospinal veins which very sensitively reflect pressure changes to that fluid. The almost constantly shifting variations in pressure which occur in the cerebrospinal veins and which are secondary to pressure changes in the extracraniovertebral veins, indicate the elastic nature of the craniovertebral cavity in contrast to the older conception of the closed box character of the cavity. Under most physiologic and many pathologic conditions, variations in cerebrospinal fluid pressure are readily explained by changes in venous pressure. It is only under unusual pathologic conditions that variations in osmotic pressure affect the level of the fluid pressure.

Although the cerebral arteries have an intrinsic nerve supply, changes in caliber of the intracranial arteries probably rarely, if ever, disturb the normal cerebrospinal fluid pressure, except during severe abnormal states, such as during oxygen-carbon dioxide disequilibrium. It is true that these vessels react sensitively to changes in metabolites circulating in the body, metabolites, however, which remain remarkably constant under physiologic conditions. Even under very unusual conditions, it is difficult to effect changes in caliber of the cerebral arteries, and great and sudden increases in arterial pressure are necessary before the diameter of the cerebral arteries alters. Again, removal of fluid from the cistern does little, if anything, to the caliber of these vessels. On the other hand, during such procedures, marked changes occur in the size and pressure of the cerebral veins.

If it is a dictum that the cerebrospinal fluid pressure is closely related to the venous pressure, it is also true that the cerebral blood flow is closely related to the arterial blood pressure and the width of the cerebral vascular bed. These two factors tend to keep the oxygenation of the brain at a steady state or within normal limits, so that, for example, a great fall in arterial pressure is compensated by a widening of the vascular bed. Thus, the blood flow may be slower, but the oxygen uptake by the brain is relatively greater. It is remarkable that unconsciousness or convulsions do not supervene until the cerebral arterial pressure falls to a level of approximately 20 mm. of mercury, at which point the oxygen saturation of the internal jugular blood is in the neighborhood of 20 volumes per cent. The difficulty in affecting the cerebral blood flow is again observed in states of increased intracranial pressure, which must be extremely high before collapse occurs. One of the most remarkable of the vascular adjustment mechanisms is the ability of the cerebral circulation to continue in the face of almost complete and prolonged venous obstruction. Under such conditions, the emissary pathways increase in their activity so that the cerebral venous pressure becomes partially adjusted, and the production-absorption mechanism becomes reestablished with a fall of cerebrospinal fluid pressure to its original level.

These examples suffice to demonstrate the remarkable resiliency of the cerebral circulation during widely adverse circumstances.

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THE INTRASPINAL (SUBARACHNOID) INJECTION OF VITAMIN B_1 FOR THE RELIEF OF INTRACTABLE PAIN, AND FOR INFLAMMATORY AND DEGENERATIVE DISEASES OF THE CENTRAL NERVOUS SYSTEM*†

PRELIMINARY REPORT

ELIAS LINCOLN STERN, M.D., F.I.C.A.

Surgeon, Department of Sympathetic Neural Surgery, Sydenham Hospital; Instructor in Anatomy, College of Physicians and Surgeons, Columbia University

NEW YORK CITY

SINCE the recent synthesis of vitamin B_1, now known as thiamin chloride,[1] considerable interest has been aroused in the therapeutic administration of this drug.[2]

That vitamin B_1 is the anti-neuritic vitamin, that its absence from the diet causes the disease beriberi which may prove fatal if untreated, that lack of this vitamin may cause inflammation (neuritis) of nerves in any part of the body with marked peripheral and central degeneration, are all accepted facts and well known. Manifold symptoms may unquestionably occur, in any combination, in vitamin B_1 deficiency, such as: anorexia, achlorhydria, depression, fatigue, lassitude, heaviness, numbness, stiffness, cramps in the muscles, anesthesia, pains, tingling sensations in the feet, edema of the ankles, limbs, and face, generalized anasarca, wasting of the limb muscles, insomnia, loss of power of locomotion, partial paralysis of muscles, foot-drop, steppage gait, breathlessness, bradycardia or palpitation, enlargement of the heart, precordial pain, and finally paralysis of the ninth and tenth cranial nerves, as well as of other sensory and motor nerves. Loss of appetite, glossitis or atrophic tongue, delayed emptying of the stomach, defective absorption in and elimination from the intestine, colitis, eructations, flatulence, biliousness, visceroptosis, epigastric distress, loss of weight, lowering of blood pressure, secondary anemia, retardation of growth, susceptibility to infection, unhealthy skin, tardy convalescence, amenorrhea or irregular menstruation, loss of libido, and sterility—all these have been at times attributed to vitamin B_1 deficiency. Headaches, hyperopia, congenital deafness, anosmia, and impotence may also be associated.

Since these may all very well be manifestations of a primary derangement of parts of the nervous system, and since post-mortem examinations in beriberi, and experimental avitaminosis B_1 have revealed marked myelin degeneration in the spinal cord as well as in the peripheral nerves,[3] the logical method of vitamin B_1 therapy appears to me to be via the intraspinal (subarachnoid) injection into the cerebrospinal fluid.

Reports have already appeared in which vitamin B_1 has proved itself of value in cases of local neuritis, sciatica, sacro-iliac, brachial, intercostal, ulnar, crural,[4,5] trigeminal, optic, auditory, etc., as well as in polyneuritis of alcoholic origin[6,7,8,9,10] or having diabetes, diphtheria, virus infection, pregnancy, anemia, heavy metal poisoning, or other disease as exciting cause. Chronic arthritis has also been

* From the Department of Anatomy, Columbia University, and the Pain Relief Clinic, Sydenham Hospital and the Manhattan General Hospital, New York City.

† Read before the Seventh Cruise-Congress of the Pan American Medical Association, Havana, Cuba, January 18–23, 1938, and before the Annual Combined Meeting of the Section in Neurology and Psychiatry and the general Medical Society of the District of Columbia, Washington, D. C., February 9, 1938.

helped.[12,13] Up to this time, however, vitamin B_1 has been administered by mouth, subcutaneously, intramuscularly, by rectum, and intravenously.[14,15,16,17] This is I believe the first report on observations when given intraspinally. Therapeutic responses, up to this time, have taken from several days to several weeks or months[2] depending upon the duration and severity of the case. The very quick response when given intraspinally is to be emphasized, as compared with the relatively slower response when given by the older methods, and some explanation for this will be given.

The cerebrospinal fluid is a clear, colorless, watery fluid, with a specific gravity of about 1.007. It bathes the brain, spinal cord, and nerve roots of the cranial and spinal nerves. It is believed that its main source is from the epithelial cells lining the choroid plexus and its blood vessels. Hassin,[18] on the other hand, believes that it originates mainly from the tissue fluids of the brain and spinal cord, and that the choroid plexus is an organ of excretion and not secretion, in so far that lipoids, blood, and inflammatory products from tubercles, abscesses and other pathologic processes may be found in large amounts in the tufts of the choroid plexus in such conditions. He also believes that no such natural filter exists in the membranes of the spinal subarachnoid space, but that foci of arachnoid cells are sometimes found there to contain such lipoids and pigment as are found in the choroid plexus. Suitable liquids injected into peripheral nerves, such as viruses, tetanus and anthrax toxins, may reach the meninges and their spaces, while dyes and lipiodol experimentally injected into the subarachnoid space have found their way out along the perineural spaces of the peripheral nerves.

Some facts concerning the circulation of the cerebrospinal fluid appear to be well established. Passing from the choroid plexus into the lateral ventricles, it proceeds by way of the foramen of Monro to the mid-ventricular system, the sylvian aqueduct and the third and fourth ventricles. From here it escapes by way of the foramina of Magendie and Luschka, and probably other apertures, into the basal cisterns which also receive the fluid from the subarachnoid space. It varies in amount from 100 to 130 c.c., and the amount in the spinal subarachnoid space is estimated at between 40 and 60 c.c. The pressure under which it flows fluctuates between 40 and 150 mm. Its rate of formation is about 2.5 c.c. every ten minutes, and it renews itself six or seven times in twenty-four hours. It has been thought to leave the subarachnoid space by way of the arachnoid villi or tufts, the dural sinuses, the veins, and the lymphatics into the general circulation. More recently, emphasis has been placed upon the rôle of the perineural spaces of the cranial and spinal nerves, as a pathway for the exit of the cerebrospinal fluid. Hassin has found that the occlusion of the perineural spaces, which occurs in pathologic conditions, such as tabes, peripachymeningitis, tubercular and leukemic, chloroma, and Hodgkin's disease, is mainly responsible for the stasis, edema, and other accompanying spinal phenomena. The changes found in the cord correspond to the identical levels of the peripachymeningitis. I wish to emphasize that obstruction of perineural spaces may block the circulation of vitamin B_1 along peripheral nerves.

While the cerebrospinal fluid may have a definite route of flow, it is possible that this flow within the subarachnoid space may be influenced by spinal injections, and that chemical solutions, such as synthetic vitamin B_1, if injected into the lumbar region will reach higher spinal regions, and even the subarachnoid space and ventricles of the brain, directly or by diffusion.

When vitamin B_1 is introduced into the lumbar subarachnoid space, and thoroughly barbitaged, the vitamin undoubtedly pervades the entire spinal subarachnoid space, and may even reach the brain. This, therefore, is a direct, topical application of a specific therapeutic agent vital for the normal functioning of nerve tissue, and so,

vital for life itself. From the subarachnoid space, the vitamin B_1 leaves by way of the perineural spaces of the spinal and cranial nerves, thus bathing the nerves which are the extensions of the spinal cord and brain, and which in vitamin B_1 deficiencies may be the seat of functional and degenerative changes. Thus it is evident that by this method, derangements of the internal glands and organs and of somatic regions can be indirectly influenced through their nerve supply.

Tislowitz[19] believes that the increase of insulin-induced hypoglycemia by vitamin B_1 is caused by an action by the vitamin on the hypophysis or the central nervous system. Conversely, we may find that lack of vitamin B_1 may be the cause of one type of hyperglycemia and diabetes. Matthews[20] states that one of the factors in the conversion of glycogen in the liver into glucose is the nervous influence of the splanchnic nerves upon that organ. Vitamin B_1 unquestionably stimulates the sympathetic nervous system when given intraspinally. From a single observation,* it would seem that the stimulation of the splanchnic nerves following the intraspinal injection of vitamin B_1 resulted in hyperglycemia during a sugar tolerance test, and that any possible effect on the hypophysis was negligible in lowering the blood sugar.

Reports of benefit to lesions of the spinal cord and brain following vitamin B_1 therapy, when given by methods other than intraspinally, have appeared. Chorea,[21] psychosis, Korsakoff's syndrome, transverse myelitis, combined tract degeneration,[22] funicular myelosis[23] are some of the conditions benefited. According to Speransky and his collaborators, under certain conditions, cerebro-spinal fluid may actually enter the brain and spinal cord along the adventitious spaces of the blood vessels in a direction opposite to the normal flow of fluid from these parts. Hence, by mixing vitamin B_1 directly with the cerebro-spinal fluid, it is not unlikely that the vitamin can actually be made to pervade the brain and spinal cord. Whether lesions of the central nervous system can be influenced favorably by this intraspinal method, and to what degree in the various conditions, remains to be proved, but so far, clinical observations point strongly to the possibility of some remarkable advantages and curative possibilities.

Just as spinal anesthetics of the cocaine series[25] and alcohol[26] have a specific affinity for the dorsal roots, so, I believe, does vitamin B_1 have an affinity for the sensory fibers. Avitaminotic nerve fibers seem to have an urgent hunger for this vitamin. This we may conclude from the prompt alleviation of pain, especially, for example, in alcoholic neuritis, and the signs of sympathetic stimulation, when vitamin B_1 is introduced into the subarachnoid space. It is highly improbable that such prompt relief could be due to metabolic (anabolic) changes in the disturbed fibers, yet this is possible because the vitamin acts as a catalytic agent which is essential for carbohydrate metabolism. It is conceivable that minute quantities of pyruvic and lactic acids may be promptly removed when the vitamin is introduced. That sympathetic fibers are also at the same time specifically stimulated, must be concluded from an analysis of the observations to be herein described. The exact modus operandi of the changes resulting from the intraspinal injection of vitamin B_1 is being investigated experimentally in animals, and we hope to report this work as soon as possible.

On the other hand, when vitamin B_1 is ingested in food or in concentrated form for therapeutic purposes, it must, to be effectual, be largely absorbed before reaching the alkaline contents of the small intestine,

* Case III. M. D. August 10, 1937. Sugar tolerance test, 100 Gm. glucose by mouth. Repeated August 11, one hour after intraspinal injection of 30,000 I.U. vitamin B_1, twelfth in her series of injections.

	August 10	August 11
Fasting blood sugar	100.0	93.8
30 min.	111.1	300.0
1 hr.	88.2	193.6
2 hrs.	83.3	98.7
3 hrs.	103.4	93.8

All urine specimens negative for sugar.

since the vitamin may be destroyed by alkalies or by proteolytic enzymes. When given by hypo, subcutaneously, intramuscularly, or intravenously, or by rectum, this alkaline barrier is much less a factor. Cases of avitaminosis have been observed even when there was a superabundance of the vitamin in the diet. The indiscriminate self administration over prolonged periods of time, of alkaline tablets and powders, and alkaline laxatives so commonly advertised is to be condemned as being harmful and likely to lay the foundation for serious avitaminotic symptoms.

That there may be a delicately balanced mechanism of supplying the nervous system with vitamin B_1 from the circulating blood in the choroid plexus has occurred to the author. As stated before, it is thought by many that the cerebrospinal fluid is mainly secreted by the choroid plexus, and it is conceivable that under certain impaired conditions as a result of inflammatory or other processes, vitamin B_1 is not filtered or diffused out and does not reach the cerebrospinal fluid in sufficient quantities to satisfy the normal requirements of the nervous system. The amount in the cerebrospinal fluid must be quickly used up. A constant supply in the cerebrospinal fluid appears essential for normal function. It is conceivable that recognized and unrecognized forms of encephalitis might be a very common cause for such changes which would prevent a normal supply from reaching distal parts of the nervous system. We may also find that mild, abortive, or unrecognized cases of poliomyelitis may actually be the cause of avitaminotic symptoms even when there is a sufficient intake of the vitamin in the diet.

In certain other cases, one may speculate that even should the vitamin reach the cerebrospinal fluid, its action may be counteracted by an abnormal alkalinity of this fluid, which in some conditions may be considerable. Hydrogen ion concentrations of 8.6 appear not to be uncommon, and this is particularly apt to be so in cases of cancer and chronic illnesses. With this possibility in view, quantitative determinations of vitamin B_1 and *p*H readings of spinal fluid are being studied in various conditions, and it will be interesting to note the changes in these figures following the various therapeutic routes of administration of this vitamin.

In determining the feasibility of intraspinal injections of synthetic vitamin B_1, some preliminary experiments were done in the Department of Anatomy, Columbia University. It was found that doses as large as 5,500 International Units (11 mg.) of synthetic vitamin B_1 could be safely introduced via cisternal puncture in adult cats without the danger of paralysis of the medullary centers or meningitis.* As a matter of fact, the vitamin when so introduced seems to have a definite stimulatory effect on respiration and the sympathetic nervous system. This action suggested the use of vitamin B_1 in conjunction with the spinal anesthetics commonly used for surgical anesthesia. This combination may lessen or even obviate the potential untoward effects which spinal anesthesia is known to have. The pathologic studies of the nervous system in normal and avitaminotic animals following intrathecal injection of vitamin B_1 is now in progress and an interesting report is hoped to be presented at a future date.

It should be emphasized here that excessive doses intraspinally may prove fatal, as demonstrated experimentally in a cat (No. 7) in whom 20,000 I.U. were injected via cisternal puncture. On coming out of the anesthesia, the animal became very irritable, thrashing around and frothing at the mouth, with rapid respirations and nystagmus, ending in generalized convulsions, twitchings of muscles, and dilated pupils. The rapid respirations were interrupted by periods of apnea about

* This "Spinalbee" is especially prepared and sterilized for intraspinal usage, without destroying its potency, and without adding any preservative which may have a deleterious effect on the nervous system. "Spinalbee" is now standardized to represent 300 I.U. per mg.

every four minutes. The animal died in fifty minutes, from what I choose to call "vitamin B_1 poisoning."

While the injection of 0.1 Gm. acetylcholine into the cisterna magna of a cat proved fatal in fifteen minutes (No. 11), the same dose mixed with 10,000 I.U. vitamin B_1 (No. 12) had no effect, and the animal is still alive after five months. Since acetylcholine stimulates the vagus and parasympathetic systems, we may surmise that the action of this drug is antagonistic to that of vitamin B_1, and may prove a life-saving remedy in accidental vitamin B_1 poisoning in man. Conversely, vitamin B_1 may prove a life saving measure in accidental acetylcholine poisoning. These observations should be remembered in view of the increasing clinical use of these two powerful and relatively new drugs.

Since the normal vitamin B_1 requirement for the adult human body is estimated at from 200 to 500 International Units per day, it was decided to try first 1,000 I.U. (2 mg. in 2 c.c. normal saline solution) intraspinally. This amount was well borne when injected into the lumbar subarachnoid space, either with or without barbitage. The dosage was thereupon increased in rapid stages until it was found that 15,000 I.U. was apt to produce undesirable symptoms, such as weakness, conjunctivitis, loss of appetite, buzzing in ears, stiffness of muscles, and slight fever, which lasted only a short time. It was also noted that while 15,000 I.U. or over sometimes caused no such inconveniences, 5,000 I.U. on occasion did. Most of the injections given were either 15,000 or 20,000 I.U. Whether these reactions occur in those cases most or least in need of the vitamin, or whether there is an individual tolerance for the drug, remains to be determined.

To date, we have failed to detect the presence of vitamin B_1 in the spinal fluids of patients with avitaminotic symptoms, or even in those without such symptoms. The chemical test used is sensitive to one part in 100,000.

When vitamin B_1 was injected into the cerebrospinal fluid, it was still found to be there several days later. We were able to detect traces of the vitamin in the urine three to four hours following intraspinal injections. On the other hand, after intravenous injection of vitamin B_1, the vitamin was found in the urine in as short a time as one hour. There is a delay in the excretion of vitamin B_1 in the urine following intraspinal injection, as compared with that after intravenous injection. After intravenous injection, the vitamin appears to be quickly eliminated in the urine, while after intraspinal injection, the elimination is found to be spread over a longer period of time. We were unable to detect vitamin B_1 in the blood serum one hour after intraspinal, or even after intravenous injection. This is probably due to the extreme dilution of the vitamin in the very large volume of circulating blood. These observations help to explain the more prompt, and hitherto undescribed effects of vitamin B_1 when given intraspinally. It appears that the intraspinal, subarachnoid injection of vitamin B_1 is probably the most direct method of administering the vitamin to the nervous system.*

Further studies of the factors regulating the rate of absorption or utilization of vitamin B_1 from the spinal fluid may help in determining the degree of avitaminosis, and be of diagnostic and prognostic value.

No clinical evidence of meningitis was ever noticed, and the spinal fluid cell counts substantiated this, since they were not appreciably increased after the injections. An interesting observation was made

* We have found that another chemical, saccharin, when introduced into the spinal subarachnoid space and thoroughly mixed with the spinal fluid, is absorbed very slowly into the general circulation. As much as 1 c.c. of 50 per cent solution (0.5 Gm.), saccharin, injected into the lumbar region of the spinal canal and thoroughly mixed with the spinal fluid, cannot at any time reach the taste buds in sufficient concentration to arouse its characteristic sweetish taste in the mouth of the patient. The same amount intravenously, in the same patient, may be tasted in six minutes, and this taste may be maintained for only fifty minutes.

in regard to the *p*H determinations. In some cases, rather high figures were reduced, as much as 0.5–0.7 following a single injection, and in one case the *p*H was brought down from 8.6 to 7 after three injections. This decrease in the hydrogen ion reading is to be expected from the nature of the drug, since the synthetic vitamin B_1 is a chloride with the formula [$C_{12}H_{17}N_4SOCl{\cdot}HCl$]. The marked clinical improvement noted in some cases may partly be due to this increase in hydrogen ion concentration.

The first occasion in which vitamin B_1 was given intraspinally was in a case of inoperable cancer of the breast with multiple metastases. The vitamin was given in conjunction with absolute alcohol-methylene blue, which up to this time I had been using alone for the control of intractable pain.[27] Following this, two cases of multiple sclerosis and one case of degeneration of the pyramidal tracts of unknown etiology were treated through the courtesy of Dr. Charles Rosenheck. The results were very encouraging in the cases of multiple sclerosis, in so far that the patients felt better, ate better, walked and talked better, had freer and steadier motion of their joints and limbs, and in one case, a very annoying diplopia almost completely disappeared in a relatively short time. With this added impetus, cases of localized neuritis of the supraorbital, lateral femoral cutaneous and sciatic nerves, as well as other cases of cancer were treated as they presented themselves. I am convinced that a large part of the pain and other characteristic symptoms which may

Fig. 1. Case III. Von Recklinghausen's disease, after eleven intraspinal (subarachnoid) injections of vitamin B_1.

Fig. 2. Back of same patient, showing numerous skin lesions in various stages.

occur in the late, or even early, stages of cancer are due to a deficiency of vitamin B_1, and probably of other vitamins. Several she always liked sweets, and in childhood, indulged in huge quantities of rock sugar. It is well-known now that a depletion of

Fig. 3. Case xxi. Intractable pruritus ani and vulvae of ten months' duration, due to vitamin B_1 deficiency established during pregnancy. Note erosions, discoloration, thickening of skin and scratch marks around anus. Controlled by intraspinal (subarachnoid) injections of vitamin B_1.

patients with cancer who received repeated intraspinal injections of vitamin B_1 maintained their general state of nutrition a surprisingly long time despite the marked and extensive progression of the metastatic growths.

A most surprising result has been observed in a case of von Recklinghausen's disease of long standing. Since the lesions in this condition are neurofibromata, and since up to this time the etiology has remained unknown, it was thought that vitamin B_1 intraspinally might have some specific effect on the lesions. (Figs. 1 and 2.) Following the very first injection, the nodules began to get softer and smaller and some even disappeared. The patient felt rejuvenated and thinks a miracle is being performed, since she has had the condition some forty-five years. An interesting point in her history is the fact that vitamin B_1 takes place very rapidly on a diet high in sugars and starches. Since the lesions are unquestionably disappearing, it is most modestly suggested that we may have found the cause for this condition and that we may now have a specific cure. Biopsies of typical nodules are being studied in the course of the treatment in the hope that some information may be obtained as to why the nodules are disappearing. Also, direct injections into the nodules were made and these were removed and examined. It appears that there is a definite change in the fibrous tissue elements of the nodules following intraspinal injection of vitamin B_1. After a number of injections, a marked decrease in the cellular elements was noted together with a large increase in the fibrous stroma. Such therapy suggests itself in fibroids of the uterus, and the varia-

tion in size which such tumors naturally undergo may well be due to changes in the vitamin B_1 intake and consumption. It may well be that retention of lactic or pyruvic acids or other chemicals may occur along nerves or at their terminal endings, which chemicals act as stimulants to nerve fibers, thus causing these growths. When vitamin B_1 is introduced into the subarachnoid space, it may possibly follow out along the nerves to neutralize these or other irritating factors.

The effects of prolonged treatment in cancer are being investigated, and in a short time, it is hoped that encouraging reports may be given. It is quite possible that these same noxious agents along nerves or at their endings, or in locations apart from nerves, may be exciting causes in some cases of cancer. In this connection

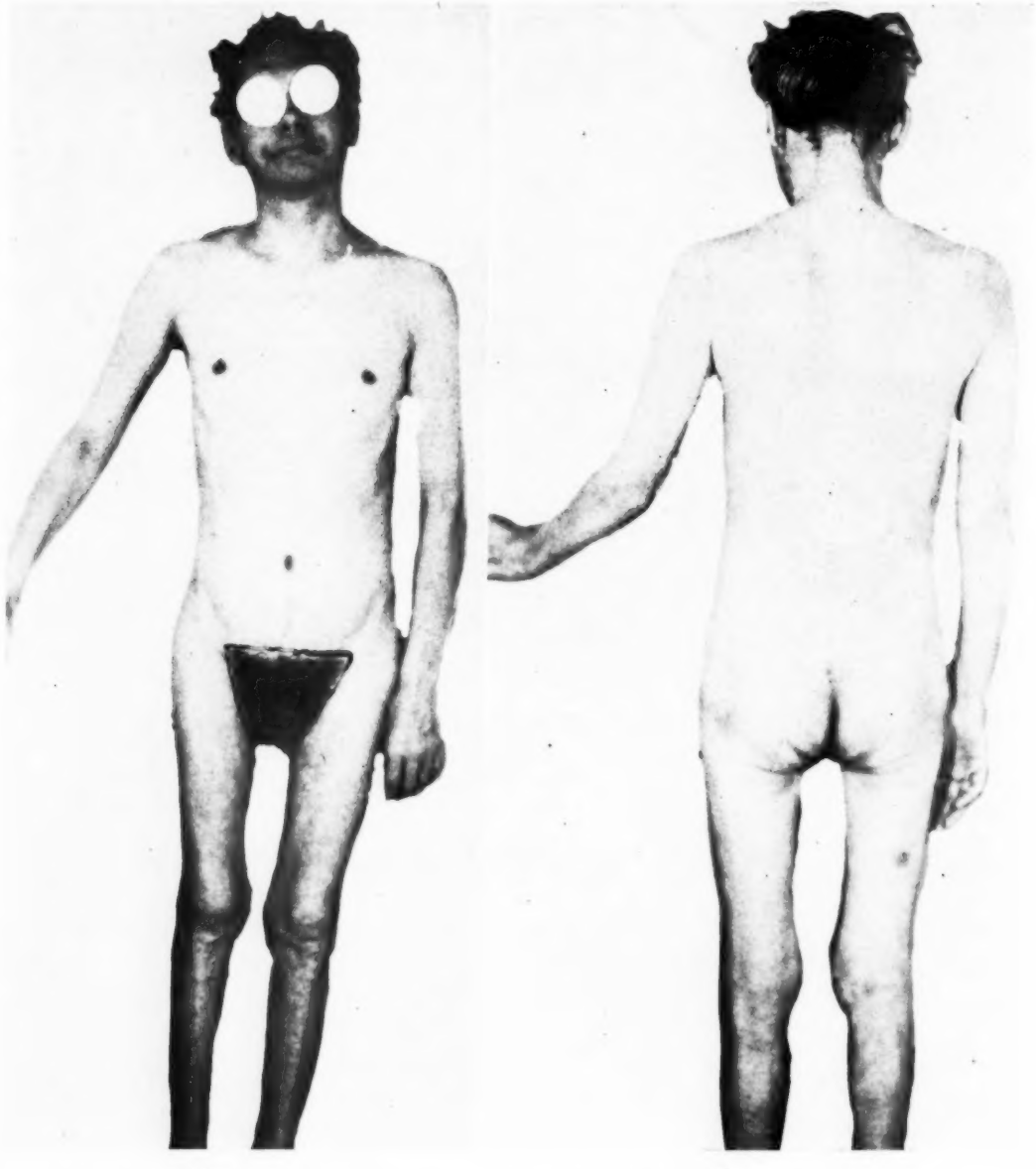

Fig. 4. Fig. 5.

Figs. 4 and 5. Case xxvi. Beriberi. Note wasting of limbs. Photographs were taken after the second intraspinal (subarachnoid) injection of vitamin B_1. The patient was able to stand and walk after having been bedridden for two months.

it is interesting to hypothecate the possible relationship and interaction of vitamin B_1 with acetylcholine which has been found along nerves and at their endings by Dale and others. In vitro, the mixture of vitamin B_1 with acetylcholine bromide, when exposed to ultraviolet rays, forms a brownish precipitate which may be related to the pigment melanin. Chemical investigations along these lines are being followed. Brief reference may be made at this point to the experimental work of Drummond[28] who found in a series of 370 rats on adequate and a similar number on inadequate vitamin B_1 diets, that there were thirteen cases of neoplasms in the deficiency group while only one appeared in the adequate group.

It has been found that somewhat better analgesic effects have been obtained in alleviating intractable pains in cancer when the intraspinal (subarachnoid) injection of absolute alcohol was preceded or accompanied by the intraspinal injection of synthetic vitamin B_1. Whether the vitamin will shorten the period of relief by aiding in the restitution of the fibers blocked by the alcohol remains to be seen. More likely, the pain symptoms will be relieved for a longer time due to the improved metabolism of the general nervous system which should follow the intraspinal injection of the vitamin B_1.

The observations of the effects of synthetic vitamin B_1 when introduced by the intraspinal route may be correlated and explained as follows:

1. The direct and immediate analgesic effect on the sensory nerve fibers in the dorsal roots with the almost spectacular relief in cases of alcoholic neuritis, may be due to the CH_2OH radical which the vitamin contains. This action would then be comparable to the analgesic effects obtained by the introduction of minute quantities of absolute alcohol into the subarachnoid space. In this connection, it is most interesting to recall a case of alcoholic neuritis of all four extremities in a patient bedridden for several weeks, in whom all pain was relieved following a single intraspinal subarachnoid injection of absolute alcohol in the lumbar region.[29]

2. Vitamin B_1 acts as a catalytic agent in the normal functioning of nerve tissue. It is an anabolic (oxidizing) agent in the restoration of functionally or organically impaired nerve tissue. Pain due to lack of vitamin B_1 may well be due to the irritation of nerves by incomplete oxidation products of carbohydrates.

3. That vitamin B_1 intraspinally acts as an immediate tonic and stimulant to vital and general functions causing a feeling of rejuvenation, may well be due to a direct stimulation of the sympathetic nervous system. Whether vitamin B_1 directly or indirectly stimulates the pituitary gland remains to be proved, but it seems most likely that the increased growth in children and experimental animals following administration of vitamin B_1 is from stimulation of the pituitary gland. The general stimulation is quite different characteristically from the stimulation which epinephrine may cause. Vitamin B_1 causes no abrupt rise in blood pressure, and the vitalizing effects of the vitamin are more prolonged.

4. The immediate effects may be due to a stimulating effect of the endocrine glands through their sympathetic nerves.

5. The general increase in metabolism may be due to the absorption of the vitamin into the general circulation, or a widespread diffusion along all the nerves in the manner described, resulting in a general increase in oxidation. That vitamin B_1 is essential for the oxidation of the degradation products of glucolysis, especially pyruvic and lactic acids has been demonstrated.[30] In a case of thromboangiitis obliterans with impending gangrene of the legs, and with normal blood sugar, sugar continued to be excreted in the urine although several injections of vitamin B_1 were given intraspinally.

6. The elimination of vitamin B_1, when introduced into the subarachnoid space, is delayed, as compared to its elimination

Case	Initials	Age	Sex	Diagnosis	1937 Date	Synthetic Vit. B1 500 I.U. = 1 mg.	Absolute Alcohol	Level	Results
1.	A. K.	53	F.	Cancer breast 3 yrs., metastases to spine, pelvis, scalp. Severe pains lower limbs.	4/16	.....	12 min.	Th12-L1	Appetite improved. Pain controlled after 2 mos. No narcotics necessary. Pain returned.
					5/5	.....	10	Th12-L1	
					5/21	1,000 I.U.	.....	L1-2	
					7/28	15,000	.....	L3-4	Followed by 10,000 U.S.P. vit. D in oil—same level.
					8/17	8,750	.....	L3-4	Followed by 3 c.c. of 10% calcium gluconate—same level. Weak spell, coarse tremors. General depression of nervous system. Pains controlled.
									12/23 Looks surprisingly well. Very little pain.
2.	D. S.	67	M.	Thromboangiitis obliterans. Pregangrenous feet; pain, swelling, discoloration, 10 mos.	5/3	.....	10	Th12-L1	Pain much relieved, swelling feet reduced, color improved, warmer. Appetite better, looked better.
					5/7	.....	9	Th12-L1	
					5/14	.....	8	L2-3	
					5/21	4,000	.....	L4-5	
					5/27	4,000	.....	L4-5	
					6/4	10,000	.....	Th12-L1	
							16	Th12-L1	
3.	M. D.	56	F.	von Recklinghausen's Dis. Multiple neurofibromatosis. 44 yrs.	5/25	1,000	.....	L4-5	Rejuvenated—feels like "18." Joints looser, walks and climbs stairs easier, sleeps well. "New motor inside." Nodules softer, smaller, some have disappeared.
					6/1	5,000	.....	L4-5	
					6/8	15,000	.....	L2-3	
					6/15	10,000	.....	L3-4	
					6/22	10,000	.....	L3-4	
					6/29	15,000	.....	L3-4	
					7/6	15,000	.....	L3-4	No desire for sweets.
					7/13	20,000	.....	L2-3	Lost 10 lbs. since start of treatment. 7/16 Vaginal bleeding few hours, first time in 3½ yrs. Menopause at 52. Bitter taste in mouth.
					7/20	25,000	.....	L4-5	Still feels like "16." Blood sugar 93.0; B.M.R. +3.
					7/27	25,000	.....	L3-4	Lost another 10 lbs.
					8/3	30,000	.....	L3-4	B.P. 150/62. Peculiar odor. See Figs. 1 and 2.
					8/11	30,000	.....	L4-5	Sugar tolerance test—see text.
					8/17	.....	.....	L3-4	2,000 I.U. vit. C. Pains in lower limbs all night—hot. Felt like after third injection. Nodules getting smaller.
					8/24	15,000	.....	L3-4	Feels well.
					8/30	.....	.....	.....	Feels like "16." Intra-muscular injection 10,000 I.U. B1.
					9/21	20,000	.....	L2-3	Best injection ever.
					9/28	20,000	.....	L2-3	
					10/6	20,000	.....	L3-4	Pains in arms and legs for one day.
					10/26	20,000	.....	L3-4	
					11/2	30,000	.....	L3-4	Pains over kidney region. Passed dark colored urine for one day. Headache.
					11/22	.....	.....	.....	20,000 I.U. B1 intramuscular. Feels like "16." No complaints. Still under treatment.
4.	J. R.	52	M.	Degeneration pyramidal tracts; etiology unknown. Difficulty in walking, holding back erect.	5/25	1,000	.....	L2-3	10 per cent improvement in gait. Feels like "2 yr. old." Weakness in legs and back continues. Eats better, looks better.
					5/28	5,000	.....	L2-3	
					6/3	10,000	.....	L2-3	
					6/7	15,000	.....	L1-2	
					6/11	10,000	.....	L3-4	
5.	S. E.	28	M.	Multiple sclerosis; difficulty walking. Pains, paresthesias. Difficulty talking.	5/25	1,000	.....	L3-4	Walks and feels better. Joints looser. Eats well. Marked general improvement. Feels "like million dollars."
					5/28	5,000	.....	L2-3	
					6/3	10,000	.....	L2-3	
					6/7	15,000	.....	L1-2	
					6/11	10,000	.....	L3-4	
6.	T. W.	32	M.	Multiple sclerosis; history of lues; negative blood and sp. fluid Wass. Disseminated lesions. Postencephalitic?	5/28	5,000	.....	L2-3	Walks and feels better; talks, sees better. Better control muscles. Increased libido, frequent erections. Joints looser. Enormous appetite. Marked improvement.
					6/3	10,000	.....	L2-3	
					6/6	15,000	.....	L1-2	
					6/11	10,000	.....	L2-3	

Case	Initials	Age	Sex	Diagnosis	1937 Date	Synthetic Vit. B_1 500 I.U. = 1 mg.	Absolute Alcohol	Level	Results
7.	W. E.	56	M.	Carcinoma rectum, op. 1932. Intractable pains sciatic neuritis 9 mos. General metastases liver, lungs, brain.	5/31	.....	8	L3-4	Slight general improvement. Pains difficult to control, central origin. Drug addiction.
					6/5	10,000	.....	Th11-12	
							10	Th11-12	
					6/11	.....	9	L4-5	
					6/18	.....	16	L3-4	
8.	J. F. G.	50	M.	Pulm. tbc. 20 yrs. Duodenal ulcer 15 yrs. Pains in heels 5 yrs., in rectum, penis and legs 5 mos., in epigastrium 3 mos. Tremor 1 leg 5 mos. Dyspnea.	6/2	5,000	14	Th7-8 L2-3	Breathing easier. Slight amelioration pain. Appetite improved; ate everything. Morphine addiction.
					6/5	10,000	16	Th7-8 Th7-8	
					6/11	10,000		L3-4	
9.	D. C.	54	M.	Alcoholic neuritis supraorbital nerve, 4 wks.	6/4	10,000	.....	L4-5	Completely relieved in 24 hrs. Further observation necessary.
10.	M. N.	40	F.	Cancer breast 3 yrs. Metastases to spine, pelvis, femur. Pains lower limbs.	6/9	5,000	12	Th11-12 Th11-12	Pains much relieved. Appetite better; looks better. Less morphine required.
					6/14	5,000	14	Th11-12 Th11-12	
					6/18	10,000	15	Th11-12 Th11-12	
					6/23	15,000	24	Th10-11 Th10-11	
					6/28	10,000	12	Th12-L1 Th12-L1	
					7/2	15,000	15	Th12-L1 Th12-L1	
					7/7	15,000	12	L3-4 L3-4	Appetite better—looks better—still has pain.
					7/30	20,000	.....	L3-4	
					8/6	20,000	16	Th11-12 Th11-12	
					8/17	.....	.....	L3-4	2 c.c. 10% calcium gluconate intraspinally. No untoward reaction. Voracious appetite.
					8/26	500	.....	L3-4	2000 I.U. vit. C combined with B_1, followed by 2 c.c. 10% Calcium gluconate. Limbs twitched and jerked—general depression.
					9/22	5,000	24	L1-2	2,000 I.U. vit. C combined with vit. B_1. Looks very well.
					9/29	.....	32	L4-5	
							16	Th11-12	
					10/27	20,000	24	Th11-12 Th11-12	Extensive cancer. Drug addiction.
11.	W. B.	49	M.	Alcoholic neuritis sciatic, lateral femoral cutaneous nerves. 6 wks. Similar attack 15 yrs. before.	6/23	15,000	.....	L4-5	Completely relieved in 24 hrs. Further observation necessary.
12.	S. Z.	43	F.	Carcinoma abdominal wall; metastasis lumbar spine.	7/15	15,000	16	Th11-12 Th11-12	Relief on left side.
					7/21	15,000	10	L2-3 L2-3	
					7/28	15,000	.....	L3-4 L3-4	Followed by 10,000 U.S.P. vit. D in oil intraspinally.
					8/4	20,000	18	Th11-12	75 per cent relieved.
								Th11-12	Died Oct. 15.
13.	R. S.	46	F.	Hypernephroma. Metastases to chest, spine, pelvis, liver.	7/16	20,000	16	Th11-12 Th11-12	Pain somewhat relieved; able to bend down. pH spinal fluid 7.3; 15 cells.
					7/20	15,000	16	Th11-12 Th11-12	Much relieved. pH spinal fluid 7.5; 20 cells.
					7/27	20,000	.....	L3-4	pH spinal fluid 7.5; 10 cells.
					7/30	20,000	.....	L3-4	Patient up and about.
					8/6	20,000	.....	L2-3	No pains at all.
					8/13	.....	.....	L3-4	Walks better; feels better.
					8/17	15,000	.....	L3-4 L3-4	2 c.c. 10 per cent calcium gluconate. Slight pains back of neck; "thrills" up and down spine. Increased libido. Followed by 3 c.c. 10 per cent calcium gluconate; weakness, pains back of neck, drowsiness 2 days.

Case	Initials	Age	Sex	Diagnosis	1937 Date	Synthetic Vit. B_1 500 I.U. = 1 mg.	Absolute Alcohol	Level	Results
					8/19	15,000	.....	L2-3	
					8/26	5,000	.....	L3-4	1 c.c. 10 per cent calcium gluconate combined with B_1; followed by 500 I.U. vit. C combined with 5,000 U.S.P. vit. D in 95 per cent alcohol. Headache for 1 hr.
					9/3	.....	16	Th11-12	
					10/13	15,000	.....	L1-2	
					10/18	20,000	12	Th12-L1 Th12-L1	Gastric hemorrhages.
					11/11	20,000	.....	L1-2	Severe anemia.
					11/16	15,000	.....	Th12-L1	Looks better.
					11/22	10,000	.....	L2-3	Combined with 5,000 U.S.P. vit. D in alcohol. Rapidly developing cachexia. Died Dec. 28.
14.	H. D.	58	M.	Inoperable carcinoma of prostate; metastases to upper thoracic spine.	8/31	15,000	.....	L3-4	Felt stronger.
					9/3	1,000	.....	L3-4	Combined with 4,000 I.U. vit. C.
					9/8	20,000	.....	L3-4	Appetite improved.
					9/18	20,000	.....	L2-3	
					9/22	30,000	.....	L3-4	Walks slightly better.
15.	J. G.	68	M.	Carcinoma of prostate; metastasis to leg.	9/4	500	24	L4-5 L4-5	2,000 I.U. vit. C combined with B_1; some relief.
					9/8	500	20	Th12-L1 Th12-L1	2,000 I.U. vit. C. combined with B_1; 12,500 U.S.P. vit. D combined with the alcohol. Much relieved.
16.	J. E.	35	M.	Chronic myocarditis and decompensation; anuria; uremia.	9/9	15,000	.....	L3-4	Water intake average 600 c.c.; output before injections were started averaged 50 c.c. per 24 hrs. After last injection output 100–200 c.c. 9/15 5,000 I.U. vit. B_1 intramuscularly; died 9/17, uremia.
					9/10	15,000	.....	L3-4	
					9/11	15,000	.....	L3-4	
17.	A. K.	65	F.	Osteoporosis of spine; question of malignancy from breast.	9/14	10,000	16	Th12-L1 Th12-L1	Slight relief on right side; plaster jacket being tried.
18.	H. S.	74	F.	Paget's disease 23 years; pains in coccyx, hips, sacrum; bedridden 3 years.	9/28	15,000	9	L4-5	Much relieved; requires less codeine. Died 10/29 cerebral thrombosis.
19.	R. B.	67	F.	Intractable pruritus ani and vulvae, dysuria, pains anterior chest, right side neck, and left upper extremity, constipation; mass left side pelvis 3 years, possible malignancy. Milk of magnesia daily for several years. Angina pectoris.	10/2	20,000	24	Th11-12 Th11-12	10/3 Itching much better; no pains in gums.
					10/6	20,000	.....	L3-4	Relieved 75 per cent.
					10/9	20,000	24	Th11-12	Much relieved. 10/25 Itching returned; angina attack. Further treatments deferred.
20.	E. C.	62	M.	Tabes dorsalis 15 years; severe lightning pains 15 years, worse past year.	10/13	15,000	16	Th12-L1 Th12-L1	Pains in legs relieved; taken off morphine; gained 8 pounds in three weeks. Drug addiction, morphine insisted upon.
21.	G. A.	32	F.	Pruritus ani and vulvae, uncontrollable, generalized pruritis 10 months. Started during last month of pregnancy; also had weakness and numbness of fingers. During entire 9 mos. vomited daily, and took 8–10 doses of alkalies daily. Definite B_1 deficiency. See Fig. 3. On verge of desperation.	10/20	15,000	.....	L3-4	No itching for ½ hr.; then burning anterior thighs. Lost 2–3 lbs. in five days.
					10/25	30,000	.....	L3-4	No itching. Burning around rectum and vulva. Contraction in lower abdomen, (uterus), like having baby. 10/26 50 per cent better. Skin looks better.
					10/28	30,000	.....	L4-5	No itching; no burning.
					11/3	20,000	16	L4-5 L4-5	No itching around rectum or vulva; slight itching across abdomen. Has to be catheterized. 11/22 No itching; bladder function now returning. Very grateful. Erosions of skin around rectum and buttocks now healed.

Case	Initials	Age	Sex	Diagnosis	1937 Date	Synthetic Vit. B_1 500 I.U. = 1 mg.	Absolute Alcohol	Level	Results
22.	W. H.	65	M.	Hypertrophic spondylitis. First intraspinal injection 1934 relieved patient for one year after having had pains left loin for 17 years. Second injection 1935 relieved patient for almost 3 years. Pain now returned.	11/8	10,000	16	Th11-12 Th11-12	Slight dull pains left, across back. Sharp pains relieved. Libido increased. Urinary stream stronger. Now able to sleep and eat well.
23.	A. B.	36	F.	Carcinoma cervix; pains both hips and lower back, one year.	11/30	7,500	9	Th12-L1	B_1 combined with alcohol and 3 min. propylene glycol. Somewhat relieved; requires less narcotics.
					12/14	20,000	8	Th12-L1 Th12-L1	Has less pain.
					12/21	20,000	8	L2-3 L2-3	Pain completely controlled.
					12/28	20,000	.....	Th11-12	
24.	B. G.	38	M.	Carcinoma rectum; inoperable. Pains in rectum, penis, lower extremities.	12/6	10,000	14	Th11-12 Th11-12	Some relief.
					12/8	15,000	4	Th11-12	B_1 and alcohol barbitaged.
							10	Th11-12	Layered.
							9	L3-4	Layered. Pains controlled.
25.	T. R.	45	F.	Carcinoma cervix; pains both hips, lower back for 1 year.	12/7	20,000	8	Th12-L1 Th12-L1	Some relief; still has pain pubic region.
					12/21	20,000	8	Th12-L1 Th12-L1	Looks better; appetite better; pains controlled.
					12/28	20,000	.....	L3-4	
26.	V. A.	43	M.	Beriberi. Loss of appetite. Polyneuritis. Enlarged heart. Enlarged liver. Atrophy of muscles. Loss of weight. Weakness. Atrophic tongue. Generalized macular and scaly eruption. Carious teeth. Low fever. Chilean—diet principally starchy. Chronic alcoholic. Bedridden two months.	12/9	20,000	.....	L2-3	pH 8.2; appetite improved. Placed on high caloric diet reinforced with vitamins.
					12/13	20,000	.....	L3-4	Pains in legs less. Burning soles of feet. Several erections—none before treatments.
					12/15	20,000	.....	L2-3	
					12/17	20,000	.....	L3-4	pH 8.4 spinal fluid, 25 cells. Able to stand up. (Figs. 4 and 5.)
					12/21	30,000	.....	L3-4	pH 8.2 spinal fluid, 25 cells. Pain only on compressing right gastrocnemius muscle. Starting to walk.
					12/23	40,000	.....	L4-5	pH 8.2 spinal fluid, 150 cells.
					12/27	50,000	.....	L3-4	pH 8.4 spinal fluid, 200 cells, before injection. 12/28 pH 8.4, 140 cells. 1/6/38. Intravenous B_1 therapy started.
27.	W. W.	28	M.	Pains around lower ribs, both sides, 3 years. Neuritis Th11 and 12 nerves. *X*-ray examination revealed floating ribs, slight arthritic changes.	12/19	20,000	12	Th10-11 Th10-11	Very much less pain on both sides. Under observation.
28.	L. McL.	4	M.	Acute poliomyelitis. Aug. 6, headache, weakness of legs. Aug. 7, pain in hips, nausea and vomiting. Aug. 8, all four extremities paralyzed. Aug. 10, neck rigid, internal convergence right eye, dull, apathetic, breathing diaphragmatic.	8/9	.....	.....	.....	Spinal fluid 654 cells, 75 per cent polys.
					8/10	.....	.....	.....	Spinal fluid 377 cells; ft. trace globulin.
					8/11	5,000	.....	L3-4	8/12, ocular signs and rigidity neck gone; brighter; eating.
					8/14	5,000	.....	Lumbar	After two injections, mentality normal; eats well.
					8/17	5,000	.....	Lumbar	Slight motion returning in fingers, forearms, and toes.
					8/20	5,000	.....	Lumbar	
					8/23	5,000	.....	Lumbar	
					8/26	5,000	.....	Lumbar	
					8/29	5,000	.....	Lumbar	
					9/1	5,000	.....	Lumbar	
					9/4	5,000	.....	Lumbar	Transferred to State Reconstruction Hospital.

in the urine after intravenous injection. When the larger doses were given intraspinally, it seemed sometimes that not all the vitamin was neutralized or utilized. The characteristic odor of vitamin B_1 was detected in the perspiration of some of the patients. Others complained of a peculiar bitter taste, or developed an annoying conjunctivitis possibly due to the excretion in the tear glands. The increased frequency of urination which occurred with many of the patients, has already been noticed by others when the vitamin had been administered by other methods. Removal of tissue fluids and reduction of edematous parts may be due to stimulation of the cortical portions of the adrenal glands. The increased amount of the cortical hormone secreted may stimulate the reabsorbing cells in the kidney tubules which might in turn result in an increased retention of NaCl in the blood. The blood would then become hypertonic to the tissue fluids and extract the water into the blood stream. Whether B_1 is likewise reabsorbed under cortin stimulation is an interesting point to be proven, since the synergistic action of vitamins C and B_1 with cortin has been demonstrated.

In addition to an improvement in certain neurologic symptoms and signs already mentioned, vitamin B_1 intraspinally has also been found to improve the cardiac reserve, reduce edema of the ankles and feet, improve the general circulation, increase the appetite, relieve constipation, increase urinary output, decrease the weight, increase libido, increase muscular tone and improve muscular efficiency, make joints more mobile, correct diplopia, clear coated tongues and improve atrophic tongues, and produce a general feeling of well being. In certain individuals, and usually with doses over 10,000 I.U., vitamin B_1 intraspinally may cause transitory weakness, loss of appetite with or without increased food consumption, frequent urination and bowel movements, marked increase in libido, tightness of muscles and stiffness of fingers, conjunctivitis, ringing, buzzing or blowing in ears, nervousness, peculiar electric sensations of hot and cold or of "activity" in the lower limbs or all over the body, slight elevation of temperature, dizziness, headache, "puffed-up" sensation in head, and frequent erections. These symptoms may last a few hours or several days.

Several cases have been reported in which large amounts of vitamin B_1 given early in poliomyelitis were followed by unusual recovery.[31] In this disease and in encephalitis, the virus enters the cells of the nervous system and often leads to degeneration of axis cylinders which may be temporary or permanent. If the cell bodies are destroyed, the axis cylinders degenerate. The destruction of the anterior horn cells in poliomyelitis appears to be similar to that produced by deficiency of vitamin B_1. The peripheral neuritis and degeneration with resulting paralysis may take months to heal and regenerate, depending upon the length of the nerves and the condition of the cell bodies. Possibly the nerve cells in children and adults on the verge of B_1 deficiency are more vulnerable to the virus. Since such cases may not be able to take food for several weeks after the onset of the disease, when the nerve cells need the vitamin most urgently, it would seem logical to administer this vitamin by intraspinal injections into the subarachnoid space, as well as by hypo and by rectum. In this manner, an additional topical and instantaneous protective mechanism is set up where most urgently needed. Recently, Jungeblut[32,33] reported that parenteral vitamin C therapy mitigates the severity of poliomyelitis in monkeys. It has been proved that vitamin C neutralizes the virus of poliomyelitis in vitro. It would therefore seem logical to administer both vitamin B_1 and C intraspinally in this dreadful disease.[34] Since the virus is believed to enter along the fibers of the olfactory nerve, it would likewise seem logical to test the prophylactic spraying of the olfactory areas in the nose with solutions containing both vitamin B_1 and C. In times of epidemics, or on

exposure to the disease, synthetic vitamin B_1 and C should be taken as a possible preventive. Its value as a possible preventive of herpes zoster after exposure to chicken-pox, is to be expected, and its administration in all virus diseases involving the nervous system should be beneficial.

Since vitamin A is also needed by the central nervous system, and since degeneration of the spinal cord, such as occurs in pernicious anemia, is thought by Mellanby to be due to a deficiency of this vitamin, it is conceivable that vitamin A alone or in combination with vitamin B_1 might be of value when injected intraspinally. And since vitamin C is markedly reduced in the brain and spinal cord in scurvy and in milder deficiencies, and has been found in subnormal amounts in the spinal fluid of chronic alcoholics, we are investigating the therapeutic value of vitamin C intraspinally. The possible value of vitamin D intraspinally is likewise being determined. To date, I have injected intraspinally into the subarachnoid space vitamins, A, B_1, C, and D, alone and in various combinations, and the detailed report of this work will appear later. It may be that a poly-vitamin solution injected into the subarachnoid space will be found to give superior results, and possible cures in baffling conditions.

SUMMARY

1. One hundred and twenty intraspinal subarachnoid injections of synthetic vitamin B_1 in doses of 500 to 50,000 international units, (1 to 100 mg.) were given to twenty-eight patients, after preliminary experiments in cats.

2. The cases treated were: inoperable cancer, 10; von Recklinghausen's disease, 1; multiple sclerosis, 2; degeneration of the pyramidal system of unknown etiology, 1; thromboangiitis obliterans with pregangrenous condition of feet, 1; duodenal ulcer with pulmonary tuberculosis, 1; alcoholic neuritis of the supraorbital nerve, 1, and of the sciatic nerve, 1; cardiac decompensation, uremia, anuria, 1; tabes dorsalis, 1; hypertrophic spondylitis, 2; osteoporosis of spine, 1; Paget's disease, 1; intractable pruritus ani and vulvae, 2; beriberi, 1; and acute poliomyelitis, 1.

3. Natural or synthetic vitamin B_1 taken by mouth, rectum, subcutaneously, intramuscularly, or intravenously may not reach the nervous system because of digestion or neutralization by the alkaline intestinal contents, or neutralization by a general alkalosis, or because of barriers in diseased choroid plexuses. Even if vitamin B_1 does reach the cerebrospinal fluid, its action may be neutralized by the alkalinity of this fluid, which may be quite high as is so often found in cases of cancer and chronic illnesses.

4. Given intraspinally, the beneficial effects appear much quicker than when given by other routes, because the vitamin remains in the cerebrospinal fluid for several days after a direct intraspinal injection.

5. After intraspinal injection of vitamin B_1, improvement in varying degrees was noted in all cases. All felt better and looked better; appetites were increased, pain was lessened or abolished completely, muscular control improved, speech and gait improved, joints became looser, libido was increased, diplopia corrected. Some of the patients felt rejuvenated.

6. The case of von Recklinghausen's disease (multiple neurofibromatosis) responded most favorably. Besides marked general improvement, some nodules became softer and smaller, and some have disappeared. The specific response to vitamin B_1 therapy suggests that the disease in this patient was due to a long-continued lack of vitamin B_1, together with an excessive amount of sweets in the diet. The possible relation of a chronic deficiency of vitamin B_1 to the cause of certain types of cancer is suggested.

7. Many chronic or incurable conditions of the central and peripheral nervous systems may respond favorably to the intraspinal subarachnoid injection of synthetic

vitamin B_1. It should prove of particular value in cases of multiple sclerosis, encephalitis, syphilis, and poliomyelitis. It should be tried in cancer cases, and whenever symptoms or signs of obscure origin fail to respond to usual methods of treatment.

8. Treatment should start with ten milligrams, and be increased every fourth to seventh day, or more frequently in urgent cases. Injections should be given intraspinally in the lumbar region and thoroughly barbitaged with the spinal fluid. Doses over 20 mg. may cause slight elevation of temperature, weakness, stiffness of muscles and fingers, loss of appetite, buzzing or blowing in ears, and conjunctivitis lasting from several hours to several days. This is usually followed by signs of improvement. The maximum single safe dose intraspinally appears to be 100 mg.

9. No patient developed any paralysis, anesthesia, or signs of meningitis following the intraspinal (subarachnoid) injection of vitamin B_1. Spinal fluid cell counts were not appreciably increased. The hydrogen ion reading of the spinal fluid was usually reduced. In all cases with a *p*H above 8.5, treatment should be pushed, but it is inadvisable to reduce the *p*H below 7.0, since restlessness and nervousness may ensue. Uncontrolled intraspinal administration may cause "vitamin B_1 poisoning," which as demonstrated experimentally, may prove fatal.

10. It is possible that synthetic vitamin B_1, when mixed with the cerebrospinal fluid, may leave the subarachnoid space via the perineural spaces of the cranial and spinal nerves. Preliminary observations point to a possible beneficial action of vitamin B_1 on diseased spinal cords and nerves when administered by injection into the subarachnoid space.

11. Introduced into the subarachnoid space, synthetic vitamin B_1 stimulates the sympathetic nervous system.

12. Synthetic vitamin B_1 is a remarkable specific oxidative and catalytic drug of great benefit to injured, diseased, or avitaminotic nerve tissue.

13. Synthetic vitamin B_1 intraspinally has a powerful rejuvenating action unknown to any other drug, and is one of the most valuable therapeutic agents at our command.

Acknowledgment is made of the helpful cooperation of Mr. Roland A. Bosee, Director of Laboratories, Endo Products, Inc., and Dr. William M. Rogers of the Department of Anatomy, Columbia University.

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A TRUE dislocation (of the spine) can only occur in the cervical region, as the oblique and vertical direction of the articular processes in the dorsal and lumbar regions respectively do not permit of dislocation without fracture.

From—"A Short Practice of Surgery" by Hamilton Bailey and R. J. McNeill Love (Lewis).

PHYSIOLOGIC INDICATIONS IN THE TREATMENT OF BRAIN INJURIES*

BENJAMIN F. DAVIS, M.D., PH.D.

General Surgeon, St. Luke's and St. Mary's Hospitals

DULUTH, MINNESOTA

IN October 1936, Barnes Woodhall of Baltimore published a review of 300 consecutive cases of acute cerebral injury admitted to the neurosurgical service of Johns Hopkins Hospital, in order to present a clinical and physiologic basis for the classification, diagnosis and therapy of such cases. Woodhall's opening sentences are somewhat caustic. "During the past decade," he states, "since the introduction of methods for cerebral dehydration, the treatment of cerebral injuries has become complicated. During this time, there has been an increasing tendency in the diagnosis of such lesions for the physiologic reaction of the organism to intracerebral pressure to be superseded by mechanical measurement of intracerebral pressure. Treatment has been guided by the degree of spinal fluid pressure as recorded on the manometer without consideration of the natural defenses of the body or the manifestations that arise when these defenses have reached their limits. Cerebral tissue that is already traumatized is further insulted by chemical solutions, or by intermittent shifts in pressure produced by drainage of the spinal fluid. Finally, the current classifications of these injuries are open to the criticism that they cannot accurately convey the extent of intracerebral damage or aid in most instances in the treatment of the damage." (Woodhall, *Arch. Surg.*, 33:560 (Oct.) 1936.)

Inspired by Woodhall's study, I have investigated the records of all the cases of brain injury treated in St. Luke's Hospital, Duluth, during the years 1934, 1935 and the first ten months of 1936, a total of 134. The body of this paper, including the classification of injuries of the brain outlined below, is based on the material found in these case histories.

In this discussion, the term "injuries of the brain" is used rather than the more common "fractures of the skull," since the former term is more specific, and puts the emphasis on that which as a rule constitutes the most important element in the clinical picture. Trauma to the head may result in serious injury of the brain without fracture of the skull, and serious or even fatal damage may be wrought by pathologic changes, traumatic in effect, arising without the application of external force as, for example, the so-called spontaneous subarachnoid hemorrhages. This standpoint is also preferable because, regardless of the cause of the injury and the coincidental disruption of other tissues, such as the bones of the skull, it is usually the affection of the brain which determines the end result. It follows, therefore, that we may reasonably classify brain injuries as follows:

1. With compound fracture of the skull.
2. With depressed fracture of the skull.
3. Without significant change in intracranial pressure.
4. With or by increased intracranial pressure not beyond the limits of physiologic compensation.
5. With or by increased intracranial pressure slowly increasing beyond the limits of physiologic compensation.
6. With physiologic collapse apparently impending.

It is to Groups 3, 4, 5 and 6 that I wish to direct special attention. A study of clinical records shows that these groups

* Presented before the Duluth Surgical Society, February, 1937.

include by far the largest number of patients. Groups 3 and 6 comprise the majority of cases, those in which there is either a minimal period of unconsciousness and a rapid restoration to normal without appreciable disturbance in other physiologic functions, or those suffering a more extensive and even fatal disruption of brain tissue without the usual signs of physiologic compensation. This group includes the two extremes of the clinical picture, the relatively mild and the very severe.

Group 4 is composed of patients in whom the period of unconsciousness may be prolonged, and in whom fever, bradycardia and possibly respiratory or vasomotor disturbances appear, but rapidly become stabilized and then show signs of improvement. Patients presenting the physiologic disturbances found in Group 4, but in whom no stabilization occurs and in whom the changes either persist without signs of relief or become more marked, comprise the fifth group.

Physiologic changes in the brain following non-penetrating trauma are due to the shock of the impact between the brain and the skull. This may result only in temporary cessation of consciousness with very little anatomic change and quick recovery. If the shock of impact is sufficiently severe, gross anatomic changes may occur, resulting in complete physiologic breakdown and death, either immediately or in a few hours. Between these two extremes lie Groups 4 and 5. In these groups, one or both of two things may happen: either the anatomic damage is sufficient to cause an inflammatory reaction with edema and swelling; or sizable blood vessels are torn and free hemorrhage occurs into the brain itself or into the spaces within the skull. In either case, the result is an increase in intracranial pressure. The tissue, lymph, and submeningeal spaces permit some swelling of the brain, but as these spaces are flattened out or are over-filled by extravasated blood or edema liquid, the expanding intracranial mass is met by the unyielding cranium. If the cause of the swelling ceases to act before the medullary centers become paralyzed by the pressure and the resulting anoxemia, the condition may become stabilized, resolution may begin and spontaneous recovery may occur. However, if the cause of the swelling continues to be active, the intracranial pressure increases to such an extent that the medullary centers can no longer function and death ensues.

The physiologic changes incident to this train of events are numerous. A few are easily demonstrable without the use of complicated apparatus or procedures and can be graphically charted, and it happens that these few changes give the key to the whole situation, so that a brief inspection of a properly kept clinical record will give a definite picture of the physiologic and therapeutic indications. No single change in itself is more than suggestive, but the general pattern of the changes is definite and conclusive. The state of consciousness is of great importance; prolonged unconsciousness, deepening coma, a lucid interval followed by unconsciousness, merit chief consideration. A pulse rate 10 or more points below the average normal, particularly if it ranges below 55, is of significance. It is said that a falling pulse rate, coincident with a rising blood pressure, is indicative of increased intracranial pressure. In our series of cases, observations on the blood pressure were not made frequently enough to verify or deny the fact. Changes in the rate, the rhythm and the depth of respiration are important, and the appearance of Cheyne-Stokes respiration is particularly significant.

The temperature should always be taken by rectum in these cases; there is usually a rise of from one to three degrees. Any sudden rise in temperature is of significance and a sustained temperature of 102 degrees F. or more occurring in conjunction with disturbances of the other physiologic functions is particularly so.

Case 1. (Fig. 1.) A child 8 years of age had suffered a brain injury with a short period of

unconsciousness, followed by rapid recovery. There was a transient rise in pulse rate and temperature, but the state of consciousness

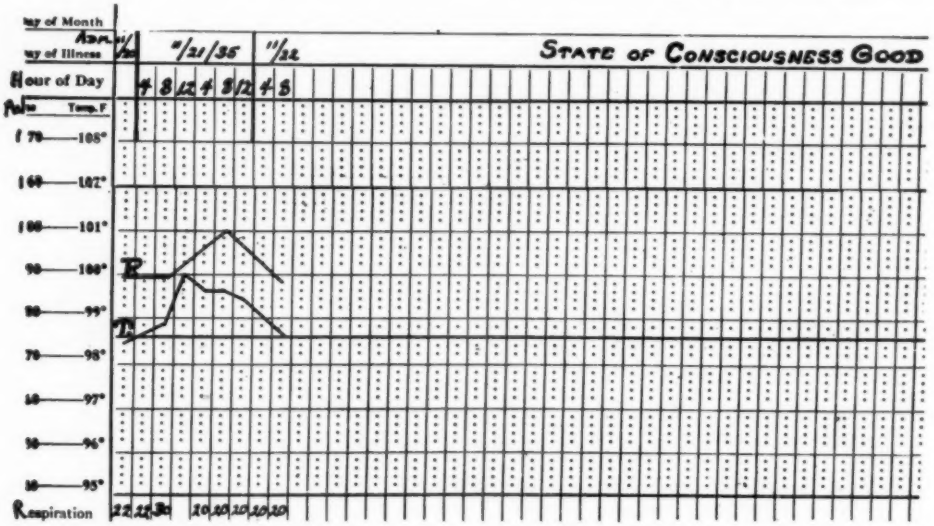

FIG. 1. Case I. Graph typical of simple concussion. Group 3.

and respiration was satisfactory. Since adults react less actively than children, the pulse and temperature curves in adults are usually flatter

eighth day after his accident, "feeling fine except for slight headache," with his state of consciousness good, his respiration and temperature normal and his pulse 56. The following morning, however, this patient returned to the hospital, complaining of severe headache and

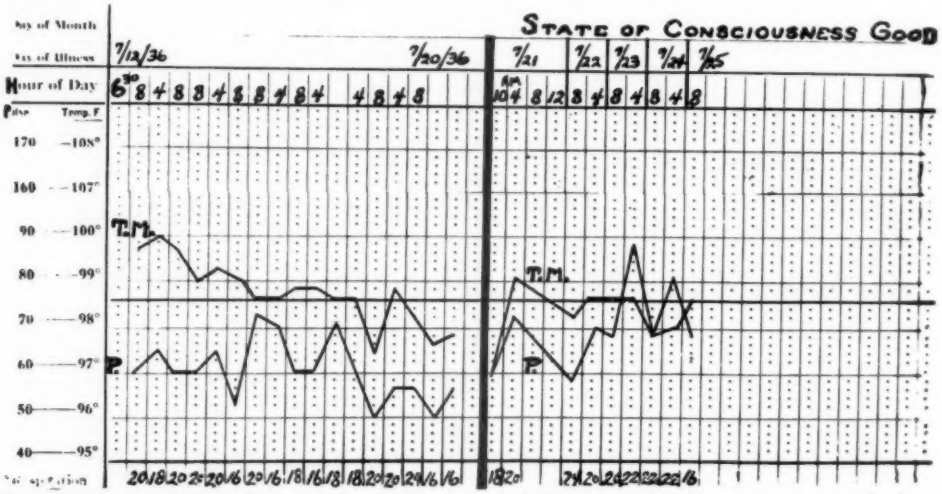

FIG. 2. Case II. Graph typical of compensated lesions with spontaneous resolution. Group 4.

than those shown here. Since recovery was rapid and complete, it is assumed that there was no increase in intracranial pressure and that damage to the brain was slight.

CASE II. (Fig. 2.) This patient fell while in an alcoholic state, and struck his head against a curbstone. He was unconscious for five minutes, but on admission to the hospital his state of consciousness was good, his respiration normal, his temperature by mouth 99.6 degrees F., and his pulse 60. He was discharged on the

vomiting. His state of consciousness, respiration and temperature were as before, and his pulse rate was 60. After thirty-six hours of rest in bed, the pulse rose to the seventies and the headache and vomiting ceased.

CASE III. (Fig. 3.) A man of 52 complained of headache and vomiting. The spinal fluid was bloody. The state of consciousness was good, respiration was normal, the temperature ranged from 99 to 101.6 degrees, and the pulse rate was 54. The pulse rate rose gradually to 70 by

the fourteenth day, and by the forty-first day had become stabilized in the low seventies, at which time the patient was apparently well and

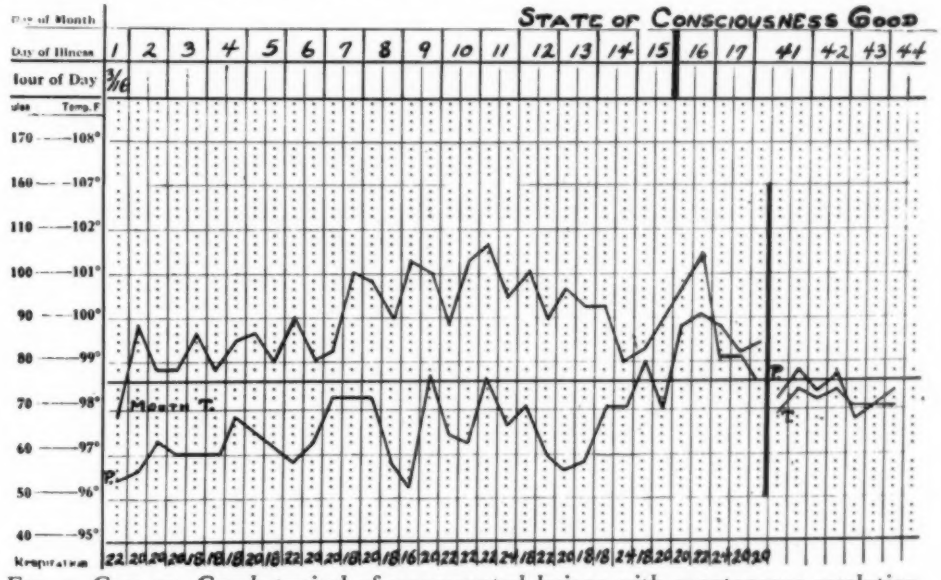

Fig. 3. Case III. Graph typical of compensated lesions with spontaneous resolution. Group 4.

his temperature normal. The clinical diagnosis was subarachnoid hemorrhage.

The patients whose charts are shown in Figures 2 and 3 are representative of the group of cases in which there is injury of the brain with or by increased intracranial pressure not beyond the limits of physiologic compensation. They serve to emphasize the fact that a pulse rate of 60 in people with brain injuries frequently is pathologic and should always, in such cases, be regarded with suspicion. The four patients whose charts are presented in Figures 4, 5, 6 and 7 are illustrations of intracranial pressure slowly increasing beyond the limits of physiologic compensation.

Case IV. (Fig. 4.) This patient had had a mild headache for one week. Then immediately after making a sudden effort, she clasped her hands to her head and complained of a sudden, severe headache. This was immediately followed by a clonic convulsion and unconsciousness. On admission to the hospital, the patient had regained consciousness; her temperature was 97.4 degrees F., her respiration 29, her pulse 64. The spinal fluid was bloody. The patient became drowsy and gradually stuporous, as the pulse dropped to 48 and the respiration to 10. Breathing became of the Cheyne-Stokes type. This condition persisted for approximately twenty-four hours when, without other change, the temperature rose to 100, the pulse to 140 and the patient died. Autopsy disclosed hemorrhage from a ruptured aneurysm of the right middle meningeal artery.

Case V. (Fig. 5.) A man, 62 years of age, was found unconscious at the foot of a stairway, with contusions over the right eye and the right temporal region. Upon admission to the hospital at 6:35 P. M., he was unconscious, with a temperature of 97.4 degrees F., a pulse of 66, and respiration 20. At 7:10 P. M., the spinal fluid was bloody and under great pressure. At 8:00 P. M., the temperature was 99.4 degrees F., pulse 48, respiration 28; at 9:10 P. M., the breathing was labored, pulse 48. At midnight, the pulse was 66, the temperature 100.8 degrees F. Unconsciousness was profound, and breathing labored. By 8:10 of the following morning, the pulse was 112, the temperature 102.4 degrees F., and patient still deeply unconscious. At 9:50 the breathing had become of the Cheyne-Stokes type. By midnight, the temperature was 106.2 degrees F., and the pulse 110 (having been 136 a few hours previously). At this time the patient died. Autopsy disclosed a comminuted fracture of the skull in the right temporal region, with rupture of the right middle meningeal artery. There was also a blood filled cavity, 3 by 4 cm. in size, in the left temporal lobe.

Case VI. (Fig. 6.) A male, age not stated, was admitted to the hospital and examined at 11:15 A. M., thirty minutes after an accident. There was bleeding from the left ear; the blood-pressure was 114/58; the extremities were normal; the pulse 48. The patient was vomiting and irrational, but not unconscious. At 11:45 A. M., a spinal tap showed that the cerebrospinal fluid pressure was 22. Following the tap, the patient roused somewhat, and his pulse rose to 64, while his blood-pressure dropped to 104/60. By 4:00 P. M., the pulse was 54, the temperature 100.0 degrees F., and respiration 28. Unconsciousness was more marked. At midnight, the pulse was 60, the temperature 102.0 degrees F., respiration 40, and the unconsciousness had deepened. At about 2:00 A. M., lumbar puncture showed the spinal fluid to be very bloody and its pressure greatly increased. The pulse rate rose to 88 at 4:00 A. M., but by 6:00 A. M., it was back to 54 and the patient was stuporous; then the pulse rate began a steady climb and at 9:10 A. M. reached 100. At this hour, respiration ceased; artificial respiration was started with the patient in the Drinker respirator. The pulse continued to beat rapidly and irregularly for three hours thereafter, after which the patient succumbed. Autopsy disclosed fracture of the base of the skull, flattening of both cerebral cortices, and laceration of the left frontal and temporal lobes.

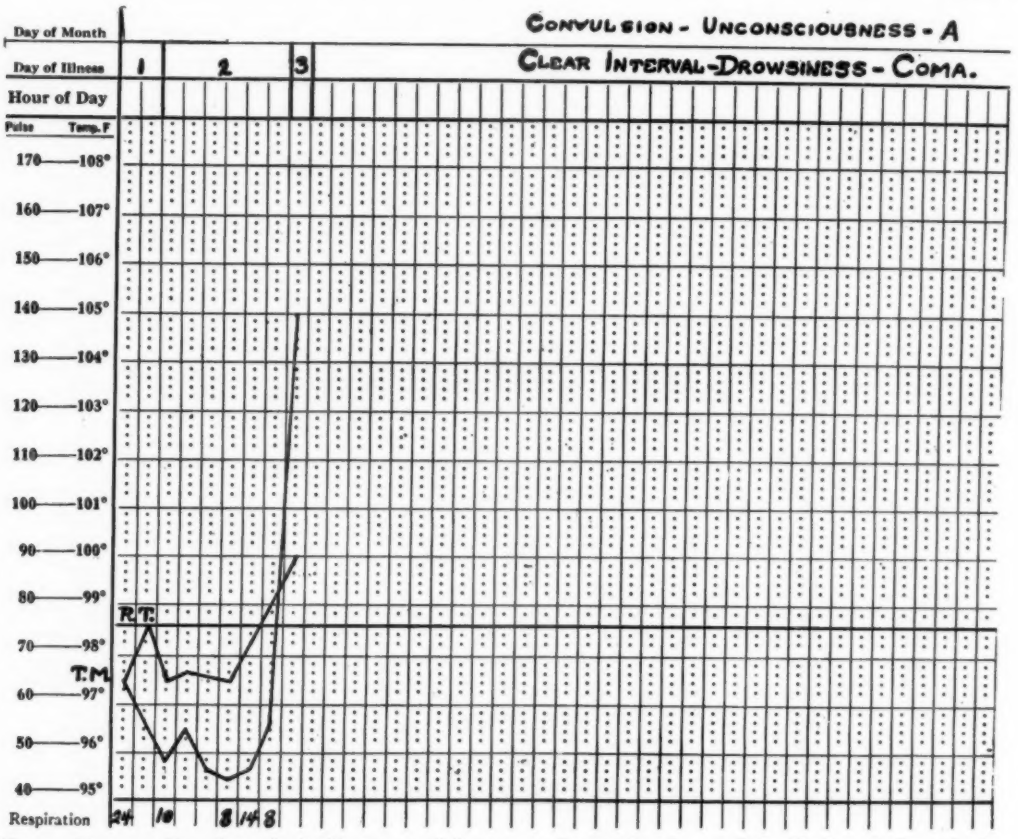

Fig. 4. Case IV. Graph typical of intracranial pressure increasing beyond the limits of physiologic compensation. Group 5.

Case VII. (Fig. 7.) A female, 27 years of age, became ill suddenly on January 3, 1935, with headache and drowsiness. She was admitted to the hospital in a conscious state on the evening of the same day with a pulse rate of 78, temperature 99.6 degrees F., respiration 22. On the following day, the pulse rate dropped to 58, while the patient became more drowsy and then unconscious. A day later the pulse rate was 56, the temperature 99.4 degrees F., and the state of consciousness less favorable; the respiration began to assume the Cheyne-Stokes type. At 4:00 P. M., an osteoplastic skull flap was turned back, the dura was widely incised and the osteoplastic flap replaced without suturing the dura. There was a temporary rise in pulse rate, but otherwise no sudden change in the physiologic reactions occurred. Within a few hours, the respirations had become

normal, the state of consciousness was improved and it was apparent that physiologic equilibrium had been established. It was not until the tenth post-operative day, however, that the pulse rate reached its normal level. The diagnosis was increased intracranial pressure, cause unknown. Less than a year later, this patient returned to the hospital to be delivered of a normal, full term baby.

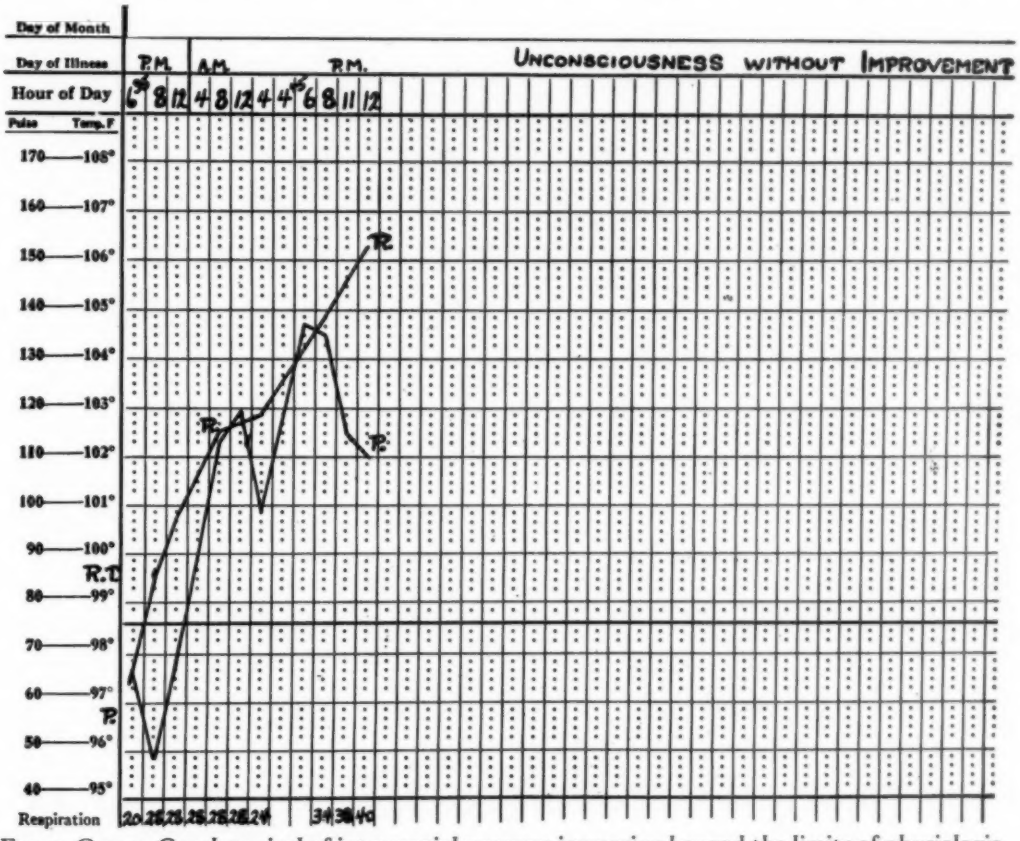

Fig. 5. Case v. Graph typical of intracranial pressure increasing beyond the limits of physiologic compensation. Group 5.

In my judgment, these four cases are typical of the type in which surgical intervention is absolutely indicated. They form Group 5 of our classification, namely, that group in which intracranial pressure is slowly increased beyond the limits of physiologic compensation. A well kept clinical record will tell the story without additional elaborate technical procedures.

Case viii. (Fig. 8.) A patient, 74 years old, was found lying unconscious at the foot of a stairway, bleeding from the left ear. Upon his admission to the hospital, his state of consciousness was bad. The rectal temperature was 102.8 degrees F., the pulse rate 88 and the respiration noisy with a rate of 28. There was involuntary urination. The patient died in about fifty hours with ample evidence of severe brain injury, but without bradycardia, and with no sign of compensation or stabilization. Instead the symptoms had become progressively worse. Autopsy revealed multiple lacerations and contusion of the brain, slight hemorrhage and edema, and a fractured skull. There was no significant increase in intracranial pressure, but damage to the brain was fatal.

Most of the charts of patients dying from cerebral hemorrhage with hemiplegia present a picture similar to that shown in Figure 8. Autopsy on several patients whose brain injury was of traumatic origin and whose clinical charts showed curves similar to this, revealed evidence of marked increase in intracranial pressure, probably sufficient to have been a factor in causing death. In some clinics, patients presenting such clinical records, not including the cases of spontaneous cerebral hemorrhage with hemiplegia, have been subjected to the operation of subtemporal decompres-

sion with apparent benefit. The death rate in the operated cases at the Johns Hopkins Hospital is 47 per cent as compared with 66 per cent among those not operated upon. It should be definitely stated that in a large number of cases the injuries are so severe that death ensues regardless of the treatment applied.

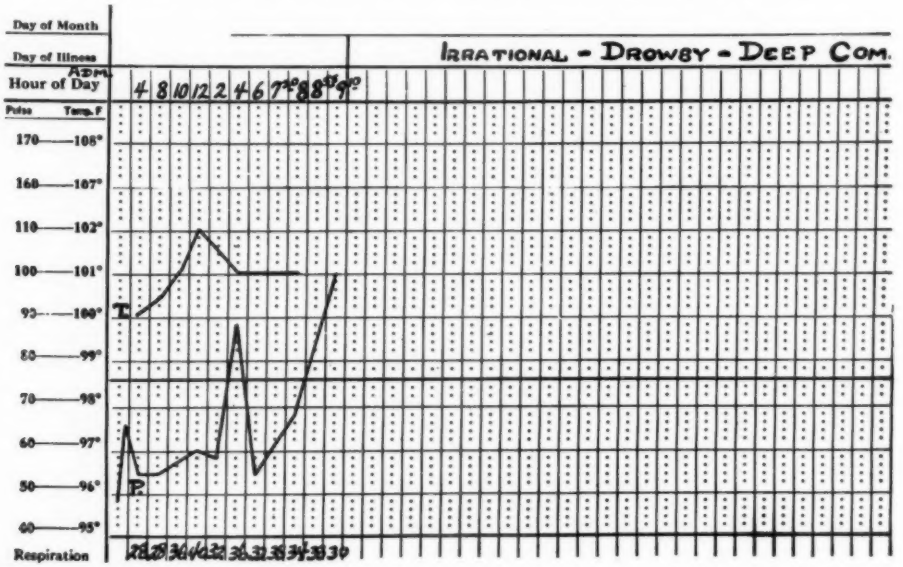

Fig. 6. Case vi. Graph typical of intracranial pressure increasing beyond the limits of physiologic compensation. Group 5.

THERAPY

In the opening paragraph of this paper, Woodhall was quoted as follows: " . . . During the past decade, . . . the treatment of cerebral injuries has become complicated." On the basis of the 134 case histories forming the source of this report, "complicated" seems a rather mild term—the picture is more suggestive of a crazy-quilt. Thus we find patients, with no physiologic indications other than transitory unconsciousness, treated elaborately with magnesium sulphate enemas, intravenous injections of hypertonic solutions, lumbar punctures and restricted diets, the doctor recording unctuously on the discharge sheet, "the patient responded beautifully to treatment." On the other hand, patients whose entire physiologic mechanisms for hours called loudly for relief from the killing intracerebral pressure, were treated by meticulously injected solutions of coramine intravenously, the Drinker respirator, hypodermoclysis, etc. Confusion runs rampant.

The answer would seem to be that the treatment of cerebral injuries is a surgical problem. The internist and neurologist will give valuable information, but the responsibility lies with the surgeon and he must make the decision as to the treatment to be given. Those of us who undertake the management of cerebral injuries must be sure that we appreciate the physiologic indications occurring in such cases and are not basing our treatment on a hodge-podge of half-digested information. We have been trying to make the patient fit the treatment and not fitting the treatment to the patient.

When a patient with an intracranial injury is admitted to the hospital, whatever else is done, we must insist on a careful record at thirty minute intervals of the pulse rate, the rectal temperature, the respiratory condition and the state of consciousness: with such a record before us, the physiologic indications will be apparent at a glance. When the intracranial pressure threatens to exceed the limits of physiologic compensation, operation should be performed at once; delay will be fatal.

Those patients who show evidence of complete physiologic collapse without preliminary compensatory bradycardia, Group 6 of our classification, are difficult to appraise. Many will die regardless of treatment. Since Woodhall's report indicates that more survive among the operated than among those treated conservatively, and since massive edema and hemorrhage many times play a large part in causing death, it is probable that nothing will be lost and something may be gained by operation in these cases. Subtemporal decompression is the operation of choice.

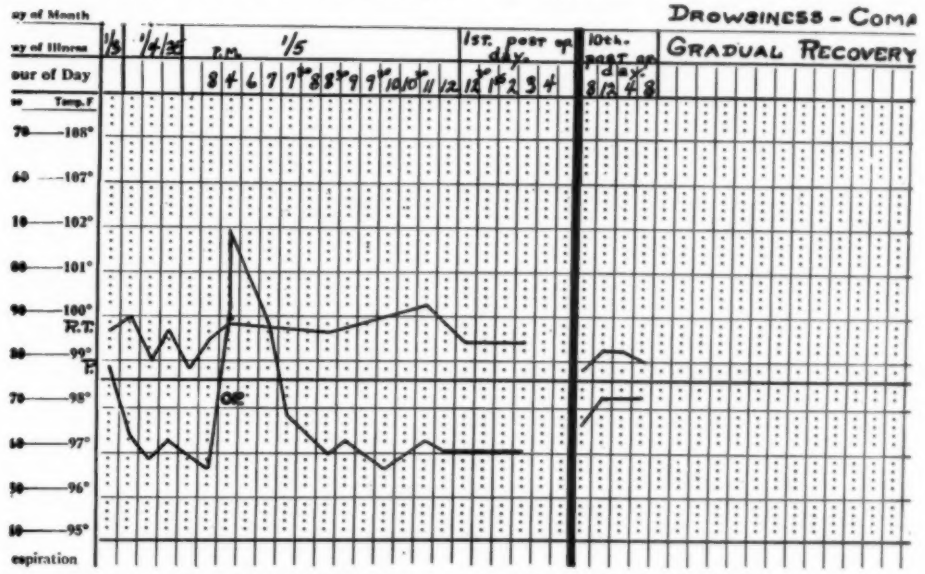

Fig. 7. Case vii. Graph typical of Group 5. Compensation established by operation.

Dehydration therapy may have definite value when used in this group of cases—I am not equipped to deny it—but my feeling is that operation offers greater chances of positive relief.

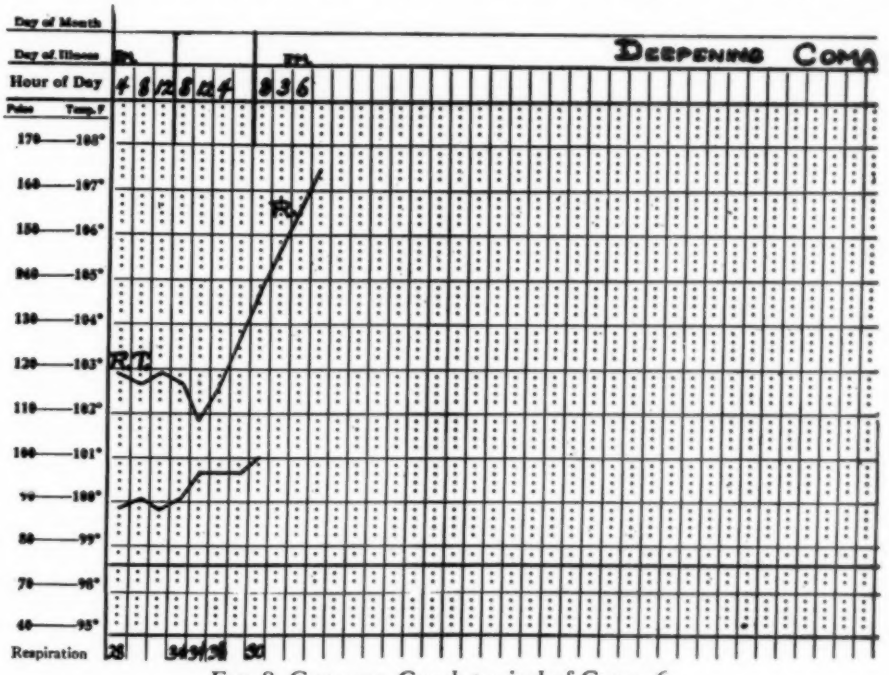

Fig. 8. Case viii. Graph typical of Group 6.

This paper is not a plea for any routine method of therapy; it is a plea for the routine study of these cases on the basis

of the obvious physiologic reactions, therapy to be based on the indications revealed by that study, and suggesting the idea that the final responsibility with regard to therapy rests upon the surgeon.

SUMMARY

A clinical classification of injuries of the brain has been suggested, according to which an estimate of the extent of the injury and of the indications for treatment may be made. Observations on the state of consciousness, the rectal temperature, the respiration and the pulse, as recorded at frequent intervals, are the keys to the classification and afford a relatively accurate index of the patient's condition. Every case of injury of the brain should be studied according to a definite, systematic plan. With such a study as a guide, the prognosis and the indications for treatment will be greatly clarified.

THERE is no doubt that one of the most dangerous lines of treatment in appendicitis is the giving of any kind of purge or even laxative. This unfortunately is too often tried with children before medical advice is called in, and probably plays a part in the rapid advance of the disease. From—"Appendicitis" by W. H. Bowen (Cambridge University Press).

GASTROSCOPY IN THE DIAGNOSIS OF GASTRIC DISEASE

HERMAN J. MOERSCH, M.D. AND ALBERT M. SNELL, M.D.

Division of Medicine, The Mayo Clinic

ROCHESTER, MINNESOTA

FOR many years there has been a constant effort on the part of the medical profession to develop instruments by which the various hollow organs of the body could be directly visualized. Such efforts have been well rewarded since the development of such methods has always been associated with an increased knowledge of the function of the organ concerned and the pathologic changes which may be present. While the interior of the stomach was one of the first portions of the body to be examined by direct visualization, the procedure was a very formidable one and did not come into general favor because of mechanical and optical difficulties. Interest in the problem lagged, for obvious reasons, after the development of the Roentgen rays as an efficient diagnostic agent.

Kussmaul,[1] in 1868, probably made the first attempt at direct visualization of the interior of the stomach; he used a sword swallower as his subject, with indifferent success. Mikulicz[2] is credited with having performed the first satisfactory gastroscopy, in 1881. Although he was successful in his effort, he apparently realized the dangers and shortcomings of his instrument as he discontinued its use. Since then, sporadic efforts have been made to improve on the gastroscope and to extend its field of usefulness. During the era of transition, the greatest advances were made by Sussman,[3] who in 1911 used an instrument which consisted of a flexible tube that could be converted into a rigid one after its introduction in the stomach, and by Schindler,[4] who in 1922 used a rigid gastroscope. This latter instrument was used at The Mayo Clinic between 1923 and 1933 and while it afforded a very excellent view of the interior of the stomach and permitted recognition of pathologic conditions in certain cases, it was not regarded as an entirely satisfactory instrument, chiefly because it afforded only a partial view of the interior of the stomach and the most important areas were not discernible in a majority of cases. Furthermore, the procedure entailed some slight risk, although when the examination was performed with care the danger was minimal. For these reasons, the use of gastroscopy as a diagnostic procedure was generally neglected until the development of the flexible gastroscope by Wolf[5] and Schindler,[6] in 1932. With this instrument it is possible to obtain a satisfactory view of the greater portion of the interior of the stomach in a high percentage of cases, and the examination can be carried out with a minimum of discomfort and risk to the patient. The technique for the introduction of the flexible gastroscope and the contraindications to its use have been so well described by Schindler[7] and by Henning[8] that further elaboration on these points seems unnecessary. The question which we propose to consider is the present value of gastroscopy as a diagnostic aid.

GASTRITIS

Gastroscopy has probably done more than any other procedure to awaken interest in this controversial subject. In spite of the interesting and comprehensive work of Faber,[9] Konjetzny,[10] and many others, the subject of gastritis has not received the attention it deserves. This is to be attributed primarily to the fact that there has been no ready method available by which the diagnosis could be established with certainty. Since gastroscopic examinations have been more frequently performed in this country and abroad, the interest

of surgeons and pathologists in the subject has been stimulated and instances of the condition have been reported with increasing frequency.

That gastritis is a real pathologic condition which deserves the greatest consideration may be gleaned from the fact that Gutzeit,[11] in his clinic, observed that chronic gastritis was twelve times as common as gastric ulcer and three times as common as duodenal ulcer. While these figures may be questioned by many physicians, they do indicate that gastritis may be a common cause of symptoms. From the standpoint of the gastroscopist, one of the most difficult problems that presents itself is to know where the borderline exists between normal gastric mucosa and that affected by gastritis. In the well marked examples of the condition, no such doubt exists and in the discussion which will follow we have concerned ourselves only with cases in which there was no reasonable doubt regarding the pathologic change.

It is not within the scope of this paper to consider the clinical features of gastritis, but rather to emphasize the value of the gastroscope as a diagnostic aid and to describe briefly the visual picture of certain common forms of the disease process. Three types of gastritis are described by most investigators, although the terminology has by no means been standardized. These types are usually designated as superficial, acute or catarrhal gastritis; chronic or hypertrophic gastritis; and atrophic gastritis. While for purposes of classification they have been divided into separate groups, the differentiation is not always clear-cut or definite, as one type of lesion may blend into the other and examples of all three types may be found in the same stomach.

The superficial, acute or "catarrhal" gastritis apparently involves only the uppermost layers of the gastric mucosa. The mucous membrane appears to be edematous and congested and there is an excess of mucus between and over the gastric folds. The process may be associated with small patches of erosion with a minimal amount of adjacent inflammatory reaction. This type of gastritis is of an evanescent nature and tends to disappear rapidly under treatment, but in some cases it may progress to the hypertrophic stage.

Chronic or hypertrophic gastritis has been divided into many subgroups but for general descriptive purposes all of these may be classified under one heading, as they blend one with another. In this type of gastritis the appearance of the mucosa may present a simple swelling and congestion of the gastric folds, which lose their normal silky appearance, or some thickening of the gastric mucosa may be apparent and mucopurulent secretion may be present between the folds. In cases in which the disease is of moderate severity, the mucosa may assume a nodular or cobblestone appearance and, if the disease is advanced, there may be marked hypertrophy of the gastric rugae, patches of ulceration and surrounding edema, infiltration, and a loss in the normal elasticity of the gastric wall. (Fig. 1.) This latter type of gastritis is a variety in which the roentgenologist is developing an increased aptitude in recognition.

Atrophic gastritis occurs with less frequency than the other forms of gastritis and is characterized by an extreme thinning of the gastric mucosa. The gastric rugae tend to disappear and the submucosal vessels, which are not normally visible, come into view. The mucous membrane has a grayish-green color, in contrast to its normal orange-red color. Jones, Benedict and Hampton[12] have described this same mucosal appearance in cases of pernicious anemia, and have demonstrated the changes that occur when liver therapy is used. While some cavil at the clinical diagnosis of gastritis in general or question the pathologic significance of the changes observed, sufficient evidence has been accumulated to establish the authenticity of the diagnosis in many cases. It has been our good fortune to observe many such

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FIG. 1. Mild degree of gastritis.
FIG. 2. Gastroscopic appearance of an acute ulcer of stomach.
FIG. 3. Gastroscopic appearance of a carcinoma near the cardiac end of the stomach.
FIG. 4. Inflammatory reaction about stoma formed by Polya type of anastomosis.

cases in which operation is performed and to have the diagnosis confirmed by microscopic examination. The present difficulty to be surmounted is obviously the proper correlation of gastroscopic, clinical and pathologic studies, and much study will be required to determine the true significance of gastritis as an independent clinical entity and its relationship to other organic or functional gastric diseases.

GASTRIC ULCER

The use of the gastroscope in the diagnosis of gastric ulcer must assume a place secondary to roentgenologic examination. However, roentgenologic diagnosis is not infallible and there is always the chance that a small ulcer may be overlooked during the course of a routine examination. Gastroscopy should always be considered in cases in which roentgenologic examination does not reveal any abnormality or in cases in which the clinical history is strongly suggestive of an intrinsic gastric lesion. It should be emphasized that gastroscopy should be considered in every case of unexplained hematemesis as it may occur from either an area of gastritis or an ulcer.

Great stress has been placed on the value of gastroscopy as an aid in following the therapeutic management of a gastric ulcer. Both Schindler[7] and Gutzeit[11] have pointed out that the healing of the ulcer as seen through the gastroscope does not keep pace with the clinical and roentgenologic improvement; visible ulceration persists long after roentgenologic examination fails to reveal any abnormality. Gutzeit was of the opinion that this latter fact is of the utmost importance in the proper treatment of gastric ulcer and in the prevention of recurrence. The greatest value of gastroscopy in this field, next to the demonstration of an ulcer, probably is the differentiation of a benign gastric ulcer and a malignant ulcer, a matter which will be considered in a later paragraph.

The appearance of a gastric ulcer on direct visualization is very striking. Such ulcers are usually found along the lesser curvature, or adjacent to it. Their appearance varies somewhat with the stage of their activity. In the acute ulcer there is usually edema, marked congestion of the surrounding mucosa, and the rim of the crater is very sharp. The base of the ulcer is usually covered with a whitish or golden-yellow débris; bleeding may occasionally be noted, although this is unusual. (Fig. 2.) In the healing and quiescent ulcer, the border is less edematous and congested than when the ulcer is in the acute phase, and the edges of mucous membrane seem to be pulled into the center, like the spokes of a wheel. Great care must be exercised to rule out a so-called phantom ulcer, which may easily be mistaken for a true one by the inexperienced gastroscopist. This phenomenon is due to a peculiar conformation of the gastric rugae, which has the appearance of a crater. It can be readily differentiated by the absence of the characteristic base of the true ulcer, while the supposed crater can be obliterated by inflation of the stomach with air.

There exist marked differences of opinion among observers as to the association and relationship of gastritis and gastric ulcer. Schindler[13] expressed the opinion that there is no relationship between the two, and noted that he had observed large ulcers without any trace of associated gastritis. Gutzeit,[11] Korbsch,[14] and Konjetzny,[10] however, emphasized the frequent association of gastritis and gastric ulcer. The exact relationship between the two is not clear, but gastritis has been thought by some investigators to be the forerunner of ulcer.

Gastroscopy is of only secondary value in the diagnosis of duodenal ulcer, unless the condition is associated with greatly retarded emptying of the stomach. When obstruction is present, a marked and definite gastritis will often be found, but gastritis is by no means a necessary or frequent finding in cases of uncomplicated duodenal ulcer.

CARCINOMA OF THE STOMACH

The diagnosis of carcinoma of the stomach in its moderately advanced stage is usually readily made by roentgenologic examination. In the early stage, however, difficulty may occasionally exist in the differential diagnosis of benign and malignant lesions. In a case of this type, gastroscopy may be of definite value in establishing the nature of the lesion, and may offer valuable information as to its probable operability. While the accuracy of roentgenologic diagnosis of carcinoma of the stomach is admittedly high, there is always a chance that a small lesion or one situated high in the stomach may be overlooked, or that marked hypertrophy of the gastric mucosa may be misinterpreted as carcinoma.

The endoscopic picture of a carcinoma of the stomach varies considerably with the nature of the growth, but it nevertheless is usually striking and can hardly be mistaken for the appearance of a benign ulcerating lesion or one of the rare benign tumors of the stomach. The appearance of a carcinoma with its blood supply intact is entirely different from the appearance of a growth that has been excised. In a carcinomatous ulcer the base of the lesion is not so clean and bright as that of a benign ulcer. The edge is irregular and ragged and local infiltration and rigidity of the gastric wall are present. Gross irregular patches of deep infiltration, marked changes in the overlying mucosal pattern, and the presence of an intraluminal mass are usually indicative of carcinoma. In association with these findings, there may be bleeding, while the grayish-white appearance of the growth itself is further substantiating evidence of carcinoma. In the diffuse non-ulcerating type of carcinoma and in lymphosarcoma, the gastroscopist may encounter as much difficulty in differential diagnosis as does the roentgenologist. This is especially true if the growth involves the entire stomach and if there is no tissue available to indicate contrast in color between normal and pathologic structures. (Fig. 3.)

Schindler[13] was of the opinion that carcinoma can be diagnosed with almost complete accuracy by gastroscopy and that it can be distinguished from benign lesions with great certainty. While there is reason to believe that this is true in most cases, we are in agreement with Henning,[8] who said that in not a few cases the final diagnosis must rest on the histologic examination. Since even experienced gastric surgeons encounter the same difficulty, this implies no great criticism of gastroscopic diagnosis.

BENIGN TUMORS AND FOREIGN BODIES

Benign tumors of the stomach may offer the greatest difficulty in differential diagnosis, even after careful roentgenologic examination of the stomach and adequate gastroscopic visualization. They are, as a rule, readily distinguished from the ordinary carcinoma by the absence of an irregular surface and surrounding infiltration. This rule does not, of course, always hold true, as it is notorious that gastric polyps may undergo secondary malignant change.

It is seldom indeed that foreign bodies in the stomach come under the scrutiny of the gastroscopist since most small and readily swallowed objects will pass the pylorus without difficulty. When this does not occur, endoscopic removal may be attempted and, if successful, gastrostomy may be avoided. In one case which was seen recently, it was possible to examine a large phytobezoar[15] through a gastroscope; the gastroscopic findings were the same as the findings at subsequent operation.

The gastroscopic appearance of syphilis of the stomach has been described by Schindler[16] and Moutier,[17] and while the condition is not infrequently seen at the clinic, no patient who has had this condition has as yet been subjected to gastroscopic examination.

POST-OPERATIVE GASTRIC LESIONS

One of the most difficult problems for the clinician, surgeon, and roentgenologist is the proper interpretation of the symptoms which may at times follow operations on the stomach. In such cases gastroscopy may be of assistance, but it is essential that the observer have knowledge of the particular type of operation performed, if he is to evaluate the findings properly. The stoma formed by a gastroenterostomy, by a Polya type of operation, or by various modifications of these procedures, usually is readily discernible. One quickly learns to recognize a properly functioning gastroenteric stoma, as it drains well, the lumen is of good caliber, and the stoma opens and closes properly. At times, it is possible to observe the rhythmic opening and closing of the stoma and pylorus in the same gastroscopic field. The jejunum is generally visible through the new stoma and can readily be distinguished from gastric mucosa by the character of its folds.

Gutzeit[11] has said that the most marked forms of gastritis are found in the stomach previously operated on; this may be true if there is evidence of obstruction, but it is equally certain that many such stomachs present an entirely normal appearance. Gastroscopy may at times be of assistance in establishing the presence or absence of a gastrojejunal ulcer. In certain cases, rather than a true anastomotic ulcer, one may find definite evidence of local gastritis and jejunitis which may account for the presenting symptoms. Such lesions are easily discernible by roentgenologic examination. Another type of lesion which may be encountered in certain cases consists of small localized regions of ulceration adjacent to the gastric side of the stoma, a condition which undoubtedly may lead to interference with proper function of the stoma. (Fig. 4.)

As Henning[8] and others have pointed out, the type of case in which post-operative symptoms develop after a gastric operation is not infrequently one in which there is visible evidence of gastritis. It is conceivable that, if all patients who were to undergo gastric operations for uncomplicated benign ulcer first were subjected to gastroscopy and that if those who had gastritis were subjected to a preliminary course of medical treatment, a decrease in post-operative complications might ensue. Paschoud,[18] who worked on this hypothesis, said that gastroscopy has been of value to him in better and more skillful surgical care of his patients who had lesions of the stomach. In any case, gastroscopy has a distinct field of usefulness in the study of complications which may arise after gastric operations, and we know of numerous diagnoses which were made by this method after other means had been exhausted. The nature of post-operative gastritis, its relation to recurrent ulcer, its occurrence, its relation to the observed symptoms, and the effects of treatment are largely unsettled problems, and it is to be expected that the gastroscopic study of post-operative complications will contribute greatly to their solution.

COMMENT

From the foregoing paragraphs it may be inferred that the principal fields in which gastroscopy is of value are many in number. In cases in which there is a conclusive history suggesting organic disease, but in which the roentgenologic findings are normal, the diagnosis should be a problem of primary importance for the gastroscopist. We have records of such cases in in which gastritis, gastric ulcer, and even carcinoma have been demonstrated and successfully treated. The second most important group comprises those cases in which there is a demonstrable gastric lesion of uncertain nature; direct visualization of the stomach also may establish a correct diagnosis in these cases. Observation of the progress of gastric lesions under medical treatment constitutes the third field; some recent experiences with patients who were suffering from benign gastric ulcer make it clear that a gastroscopic

examination of supposedly "cured" patients may not be amiss. Finally, in cases in which there are persistent post-operative symptoms but the results of roentgenologic examination are negative or inconclusive, the patients should not be dismissed from consideration or told that their symptoms are of functional origin until gastroscopic examination has been performed.

Schindler[13] said that gastroscopy is of such value that it should be used as a routine procedure in all gastric examinations. Whether time will justify such a view is doubtful; there is no doubt, however, that direct visualization of the interior of the stomach should be attempted in any case in which clinical and roentgenoscopic evidence is inconclusive or of a negative nature, but in which there is symptomatic evidence of intrinsic gastric disease. Gastroscopy does not replace careful clinical or roentgenologic study, but is rather an adjunct to diagnosis and therapy. Used in this way, gastroscopy has a place in the diagnosis of lesions of the upper digestive tract.

SUMMARY

Gastroscopy is a procedure which should be an adjunct to the clinical and roentgenologic study of selected cases of known or suspected gastric disease. Direct visualization of the stomach is of value in the examination in cases in which the clinical and roentgenoscopic evidence of gastric disease is uncertain or absent. It is particularly useful in those cases in which symptoms persist following an operation on the stomach. It has stimulated renewed interest in the problem of gastritis, and thereby has increased the interest of clinicians and roentgenologists in an effort to develop criteria by which they may determine the presence of such lesions with greater accuracy. Further studies must be carried out to determine the significance, importance, and relationship of gastritis to other clinical and pathologic findings; this is a field in which gastroscopy may be of paramount importance.

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GASTRIC CARCINOMA

D. PHILIP MACGUIRE, A.B., M.D., F.A.C.S.

Assistant Clinical Professor of Surgery, New York Post-Graduate Medical School and Hospital, Columbia University

NEW YORK CITY

APPROXIMATELY fifty per cent of the clinical admissions for gastric malignancies are inoperable, owing to the presence of Virchow's nodes, the supraclavicular lymph glands, and Blumer's shelf. Peritoneal involvement of Douglas pouch and rectal shelves, and positive evidence of hepatic and umbilical regional metastases, demonstrated by the presence of fluid and metastatic masses, also prohibit operative procedure. From the Roentgen standpoint, an involvement of the pars cardia is definitely inoperable, and that of the pars pylorica with the pars media is also inoperable in a certain percentage of cases.

Twenty-five per cent of deaths from cancer are due to gastric carcinoma. The exact site of the tumor is the same to-day as was given in Welch's summary[1] which is briefly as follows: the pyloric region (1a), lesser curvature (1b), cardia (1c), posterior wall (1d), involving the greater part of the stomach (1e), the greater curvature (1f), anterior wall (1g), and the fundus (1h). Multiple tumors are also occasionally discovered.

Carcinoma of the stomach may be present in various forms, and may vary in size; some are flat, rounded or lobulated growths that project from the inner surface. The overlying mucous membrane may be intact or ulcerated and the destructive process advanced to a degree so that the ragged or smooth edged ulcers attain considerable size. The stomach wall may become involved, peritoneal adhesions may form and perforation occur. However, carcinoma may assume the form of a diffuse infiltration of the stomach wall. This type of growth is less liable to ulcerate and is quite often overlooked or mistaken for a chronic inflammatory lesion. The most common variety is the adenocarcinoma which metastasizes early, and is characterized by distant metastases. Scirrhous carcinoma is frequently found and is usually of the infiltrating variety. A third type, gelatinous carcinoma, often forms extensive growths and is very apt to invade adjacent structures, establishing metastases. The most common secondary complications are stenosis, adhesions, fistulae, distortion, perforation and hemorrhage.

The roentgenologist classifies gastric carcinoma as to its form, location and extent. Carcinoma of the stomach invariably originates in the mucous layer, and, while basically they are all epithelial neoplasms, they present numerous structural differences.

Radiologists recognize three forms, viz.:

1. The fungous type, almost wholly epithelial in form, hence quite soft. This variety includes the adenocarcinomas, cauliflower and medullary.

2. The scirrhous type, infiltrating in form, and characterized by a relatively large amount of connective tissue.

3. The colloid type, the degenerative mucoid variety.

Lymphatics of the Stomach. In his Thèse de Paris of 1900, Cuneó described three principal groups of perigastric lymph nodes: (1) nodes of the cardia and lesser curvature; (2) the hepatic (pyloric) chain; and (3) the splenic group. At autopsy, he found an invasion of the perigastric, diaphragmatic or retroperitoneal nodes in 85 per cent of the cases. Extension to the thoracic nodes is not uncommon. The subhepatic and pancreatic nodes are among the first to become involved, as well as the mesenteric nodes and the lumbar and

aortic trunks, which also affect the inguinal nodes.

The importance of the enlargement of the supraclavicular nodes of Virchow in the diagnosis of gastric carcinoma was first emphasized by Troisier.[2] Hechler[3] introduced the important fact that Virchow's nodes were not always true metastatic carcinoma, but might also be inflammatory in nature. The left nodes are most frequently affected, but either or both sides may become involved according to the anastomoses of the thoracic duct. The invasion penetrates the thoracic duct, which may become infiltrated, resulting in a chylous ascites. This condition has a greater predilection for males than females, a predominance even more pronounced in Germany than in this country. Out of 312 cases at the University of Göttingen reported by the pathologist, Professor Edward Kaufmann,[4] 226 (72 per cent) were men.

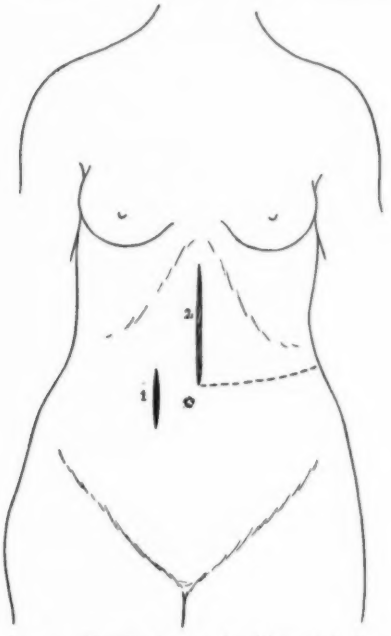

Fig. 1. Preliminary incision (1) for jejunostomy and alimentation; (2) for gastrectomy.

Metastases in the adjacent lymph nodes, viz., epigastric, portal retroperitoneal, and the more distant supraclavicular, are quite common. Numerous observers have discovered a primary isolated carcinoma with a large mass of nodes. The liver becomes involved in approximately a third of the cases.

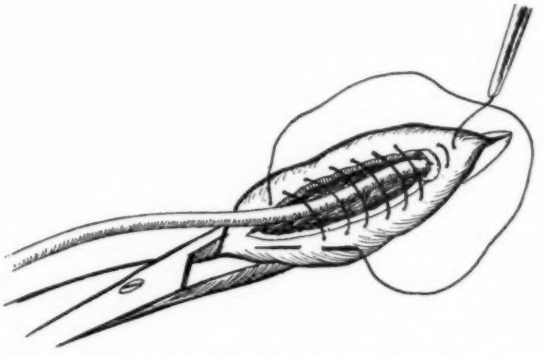

Fig. 2. First stage of jejunostomy. Introduction of catheter into the lumen of jejunum and beginning the fixation of catheter to the wall of the jejunum.

Gastric carcinomas grow with varying rapidity; the firmer variety forms more slowly, and usually the more cellular and softer ones more rapidly. The carcinoma first infiltrates the mucosa, then the muscularis, which generally hypertrophies and finally atrophies due to an increased invasion. The tumor gradually reaches the serosa from the muscularis.

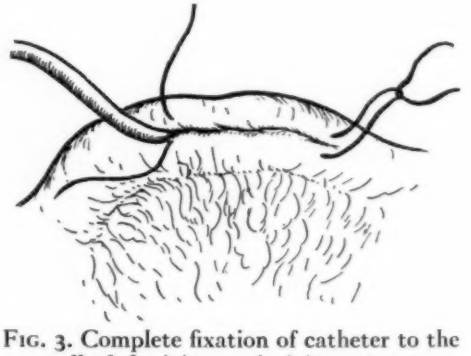

Fig. 3. Complete fixation of catheter to the wall of the jejunum in jejunostomy.

A disturbance in the production of hydrochloric acid is noted and also a fermentative process developed under the influence of various micro-organisms, especially yeast, fungi, and also bacteria. Alkalosis and a fall in chlorides, together with a rise in non-protein nitrogen and calcium, are also frequently found in the blood chemistry determinations of those cases in which pyloric obstruction occurs. At the present time, however, there is a

patent disregard for chemical analysis of the stomach for either diagnostic or prognostic purposes.

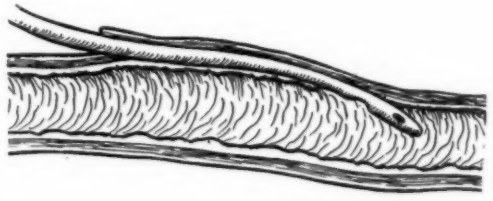

Fig. 4. A, cross section showing French soft catheter in the lumen of the gut.

Many operators, with considerable experience in this surgical field, have repeatedly become enthusiastic about new diagnostic tests which afterwards have invariably proved disappointing; the roentgenologic examination has ultimately been accepted as the only safe guide. Surgeons who believe that one gastrointestinal series is sufficient, are cautioned to have their patients return for later check-up when the readings may be entirely different. The laxity of the laity in seeking proper medical advice is second only to that of some of the doctors of medicine who are quite often even worse offenders.

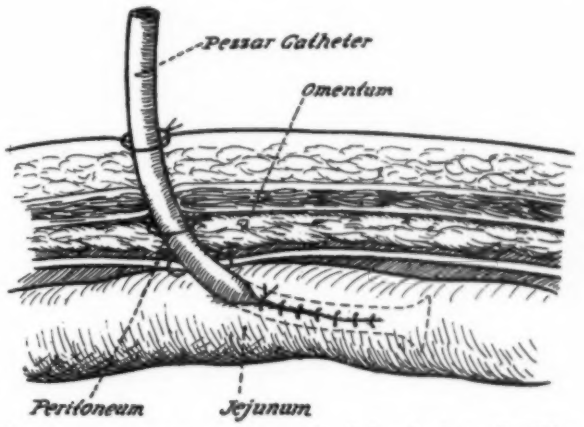

Fig. 4. B, cross section of abdominal wall. The peritoneum attached by fixation suture to the jejunum through a large opening in the omentum, and the Pezzer catheter or French catheter in the intestinal lumen.

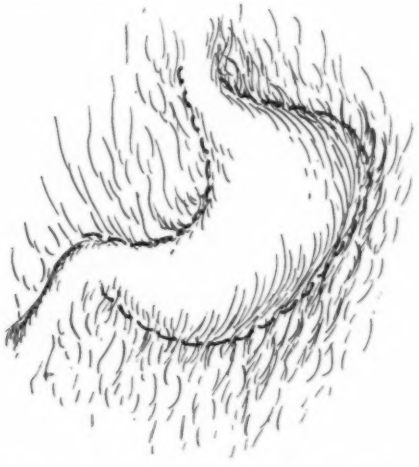

Fig. 5. Resection of gastrohepatic and gastrocolic layers with their contained blood vessels.

The primary importance of pre-operative preparation cannot be over-emphasized. There should be constant group consultations, particularly between the internist and blood chemist, and it is advisable to utilize the more improved tests for daily blood chemistry determinations and urinalysis. Three tests that have proved most accurate for the determination of kidney dysfunction or renal efficiency are: (1) the Lashmet and Newburgh method for concentrating ability; (2) the urea clearance test of Van Slyke; and (3) the fifteen minute phenolsulphonephthalein excretion technique of Chapman and Halstead to measure the efficiency of dye elimination. Alkalosis, chloride deficiencies, and calcium and non-protein nitrogen, should be watched carefully in the blood chemistry determinations. Liver dysfunction and pancreatic disturbances can be early de-

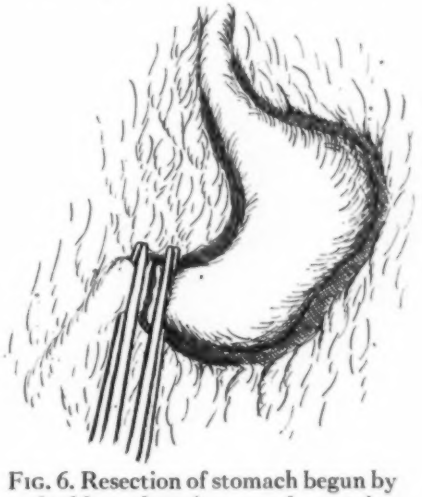

Fig. 6. Resection of stomach begun by double clamping and cutting through the duodenum by cautery.

tected by the blood chemists. Therefore, careful observation for the presence of alkalosis, chloride deficiency, kidney dysfunction, abnormal calcium percentages, liver and pancreatic dysfunctions, should be maintained in these cases.

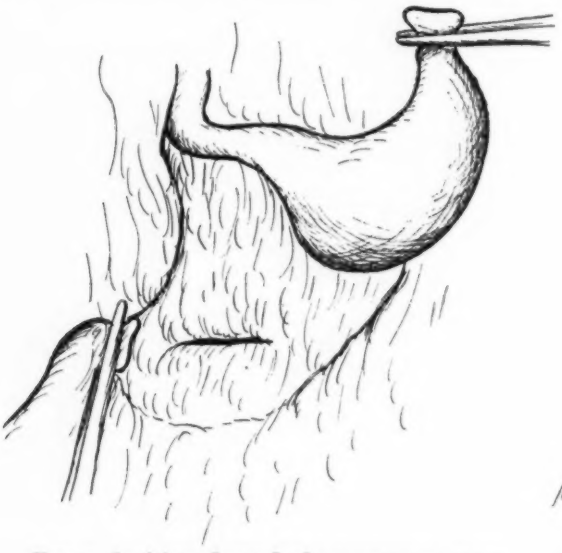

Fig. 7. Incision through the transverse mesocolon and elevation of the stomach to the left side.

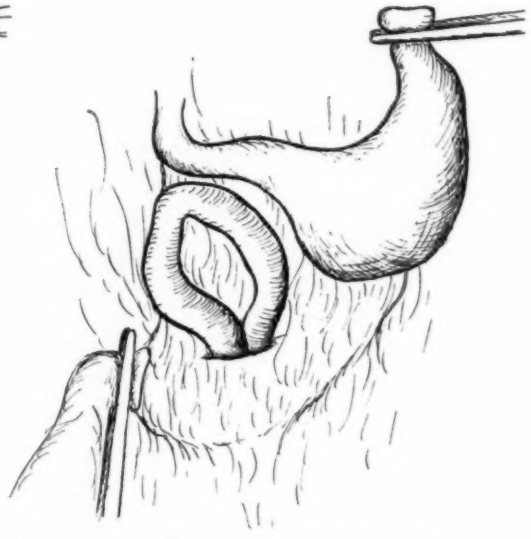

Fig. 8. Loop of jejunum carried through opening in transverse mesocolon.

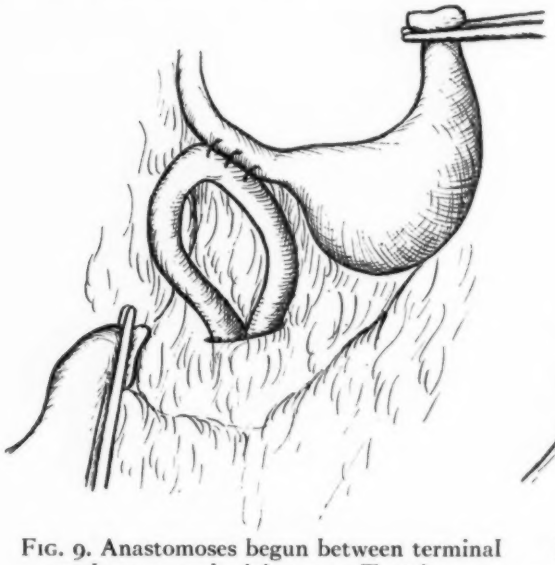

Fig. 9. Anastomoses begun between terminal esophagus and jejunum. Traction on stomach by clamp.

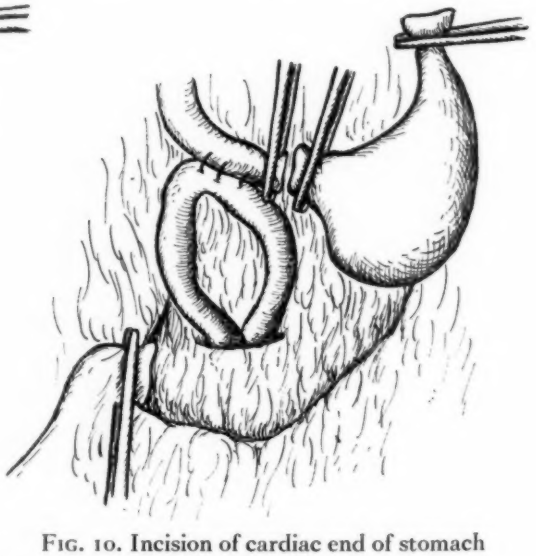

Fig. 10. Incision of cardiac end of stomach between clamps.

In every malignancy of the gastrointestinal tract, especially the ulcerating types, a toxemia develops which is mild at first, but becomes progressively severe, proportionate to the amount of open, absorptive surface. This condition is quickly reflected in the disturbed daily blood chemistry determinations. Consequently, the toxemia alone requires the administration of transfusions even in the mild cases, and massive transfusions in the more severe ones. The continuous intravenous injection of dextrose or glucose, (5 per cent), utilizing the Herndon, Tauroff or Lindeman needle, or the 10 per cent solution given by the Murphy drip or clysis method allays the alkalosis. Chloride and calcium deficiencies can

be controlled by intravenous injections and oral administrations. There can be no question that the preferable types of

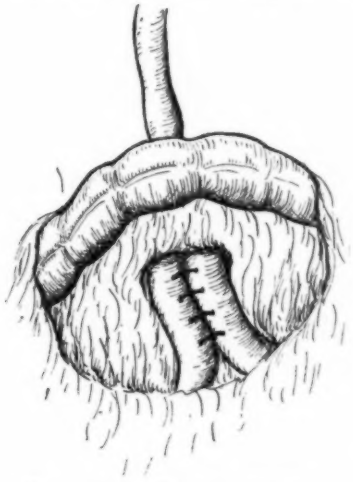

Fig. 11. Entero-enterostomy, joining loops of jejunum.

procedure consist of the oral administration of tap-water or, should this be inadvisable, the continuous duodenal drip method employed by Joseph McCarthy, Director of the Urological Division at the New York Post-Graduate Medical School, and his associates. Killian and Kast of our hospital staff, in 1918, also observed a changed blood chemistry in abdominal malignancies.

Since 65 per cent of the body weight is composed of water, a patient weighing 100 Kg. would have a water content of 65 Kg. or 6,500 c.c. A 10 per cent loss of this body fluid would prove fatal. Consequently, the intake and output of fluid is an essential in the proper determination of the blood chemistry.

During the routine preparation of the patient, biopsies should be made of the supraclavicular or other nodes.

For carcinoma limited to the stomach or metastases considered controlable, the radical continental technique, exemplified by the Hoffmeister-Finsterer, von Haberer or von Eiselsberg operative procedures, is advisable. Block abdominal anesthesia, cyclopropane, ethylene, spinal or diluted amounts of avertin should be used for a primary anesthetic.

When a malignancy invades a considerable amount of gastric area, which is well illustrated in cases involving the pars

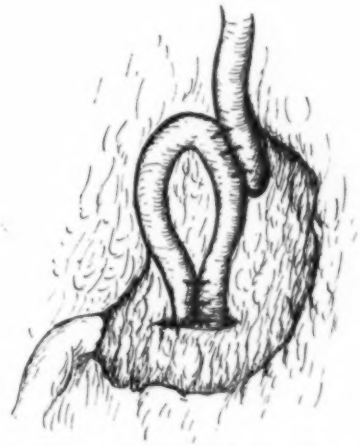

Fig. 12. Completed anastomoses of terminal esophagus and jejunal loop. Entero-enterostomy and closure of opened duodenal stump and esophageal stump.

pylorica, pars media, and also those of linitis plastica complicated by ulcers, or ulcers affecting the pars cordica, a total gastrectomy should be considered. The author believes that his method of operative procedure is particularly amenable for this condition. It consists in performing a jejunostomy through a separate small incision (Fig. 1), as a preliminary step, supplemented by the administration of the Scott-Ivy diet,[5,6] according to the method introduced by John Wolfer.[7]

The incision should be made well on the side of the median line so as not to interfere with the subsequent secondary procedure. Utilizing either the Witzel technique of the Coffey modification, a catheter should be inserted for 20 to 25 cm. The author prefers the pezzer catheter, with the perforated tip cut so that only a shoulder remains. (Fig. 2.)

When selecting the site for jejunostomy, the operator should allow 45 to 50 cm. for the anastomosis with the esophagus, so that it is advisable to choose a location 60 cm. below the duodenojejunal angle. It

is necessary to devise a support for the anastomosis by suturing this portion, between the terminal esophagus and the jejunal loop, to the peritoneum under the diaphragm, and if possible, to the edge of the suspensory ligament of the liver. This step is important since the movements of the diaphragm might tear the sutures for the anastomosis if it lacks support. Another essential detail is that the catheter should extend from 20 to 25 cm. into the lumen of the jejunum. Jejunal alimentation may also be accomplished with the aid of an electrically driven pump mentioned by John A. Wolfer.[7]

CONCLUSIONS

Fluid intake is immediately diminished when the dysfunctions have been successfully treated, as indicated by the favorable blood chemistry determinations.

Roentgenologists can make an accurate diagnosis in 95 per cent of gastric carcinoma cases. Their advice regarding the inoperability of many of these cases is of particular importance.

Fungoid carcinoma produces multiple, irregular filling defects, and the actual extent of the malignancy is quite closely delineated by the Roentgen shadow; while the scirrhous variety causes a less gross filling effect, which may gradually taper off and not indicate the actual limits of the disease. Disappointments are, therefore, quite common concerning this latter type, since subsequent exploration frequently reveals an inoperable condition contrary to the condition indicated by the *x*-ray films prior to operation.

Included in the work-up of these cases, is routine *x*-ray of the lungs and bones, supplemented by a careful survey of these bones to determine the possible existence of a deforming arch of the diaphragm due to the presence of nodular metastases.

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THE THERAPEUTIC VALUE OF GLYCERYL TRINITRATE IN BILIARY COLIC AND IN THE POST-OPERATIVE PHASE OF BILIARY TRACT DISEASE

R. Russell Best, M.D., F.A.C.S. and N. Frederick Hicken, M.D.

Assistant Professor of Surgery and Anatomy, University of Nebraska College of Medicine

Instructor in Surgery, University of Nebraska College of Medicine

OMAHA, NEBRASKA

At the meeting of the American Medical Association in Kansas City, in 1936, we presented our studies on spasm of the lower end of the common bile duct, dwelling upon its presence as a cause of some apparent operative failures. Certain therapeutic measures which for years have been advocated for their relaxing effect upon smooth muscle were also discussed. Cholangiographic studies of patients proved that the much used combination of morphine and atropine at times had not released the spasm of the sphincter of Oddi and thus had not mitigated their distress. We introduced the use of glyceryl trinitrate tablets as a means of relaxing spasm of the lower end of the common duct or sphincter of Oddi and were able to demonstrate their effect by cholangiographic visualizations. The results were particularly interesting in view of the fact that the glyceryl trinitrate had brought relief after several other drugs, including atropine and morphine, had failed.

Although we had been investigating for some time the effects of various drugs and foods on the sphincter muscle, our introduction to glyceryl trinitrate came from the casual remark of a patient who had continued to experience occasional attacks of colic after the removal of his gall-bladder. He was of the opinion that the tablets which he used for typical angina attacks also relieved him of his indigestion. This led us to experiment with glyceryl trinitrate in certain patients in whom spastic biliary dyssynergia was manifest, and who were carrying common duct tubes or catheters. We were able to observe the antispasmodic effect of this drug by cholangiograms. The alleviation of any associated distention or distress suggested its clinical use in all types of biliary colic.

In almost any phase of medicine or surgery, an attempt to release spasm which is resulting in distress or malfunction becomes a paramount therapeutic endeavor. One could enumerate endlessly such clinical entities as ocular spasm, bronchial spasm, coronary spasm, pyloric spasm and anal sphincter spasm. Here the clinician's attention is directed to relieving the contraction by drugs, application of heat, rest, or stretching the spastic muscle. In the last few years, definite advances have been made in surgical attacks upon the sympathetics and parasympathetics for relief of muscle spasm or spastic types of dysfunction.

That certain parts of the biliary tract are capable of arriving at a similar state is more clearly understood if we review some of the anatomic features. Compared with other hollow visci which empty their contents by muscular contraction, the gall-bladder is relatively thin-walled; yet in contrast to the remainder of the extra-hepatic bile tract, with the exception of the lower end of the common duct where there is an apparent sphincter, there is a preponderance of muscle tissue. During the last two years, working in conjunction with Dr. John Latta, histologist at the University of Nebraska, we have made histologic studies of the gall-bladder, cystic duct, hepatic ducts, and common duct, in an attempt to determine the comparative amounts of fibro-elastic and muscle tissue. The gall-bladder wall consists of muscle tissue which is more abundant in the

fundus and neck of the gall-bladder and diminishes as the cystic duct approaches the common duct. Some investigators, for example Luetkens[8] from Oschoff's laboratory, report the existence of a sphincteric structure at the junction of the gall-bladder with the cystic duct in 75 per cent of human subjects. According to Ivy,[7] Volborth, after making observations on dogs with biliary and duodenal fistulae, assumed the presence of a sphincteric mechanism somewhere between the common duct and the gall-bladder, which is called the "collum cysticus" sphincter. We were unable to establish this physiologic sphincter as a definite entity, but certainly would not deny its presence. We have been able, however, to verify the observation of other investigators, including Hendrickson, Oschoff, Berg and Nuboer, that the hepatic and common bile ducts down to the region of the ampulla and the sphincter of Oddi, consist mainly of fibro-elastic tissue with only scattered strands of muscle tissue. At the lower end of the common duct, non-striated muscle again appears and here reaches its maximum thickness, forming a sphincter entity which mechanically guards the entrance into the duodenum, and which, clinically, induces syndromes typical of sphincteric disturbances elsewhere in the body.

Our appreciation of the nervous mechanism of the gall-bladder would be much simpler if the parasympathetic nerve trunk carried only fibers of the motor group and the sympathetics (splanchnics) carried just the inhibiting fibers. The number of activating and inhibitory fibers in either nerve group probably differs with the individual, which would account for the apparent discrepancies in the action of various drugs upon non-striated muscle tissue. In terms of extrahepatic biliary tract innervation, the complication of reciprocal innervation enters, as we find that stimulation causes the motor fibers of the vagus to contract the gall-bladder and relax the choledochal sphincter, both non-striated muscle structures, and, conversely, causes the splanchnics with their inhibitory fibers to relax the gall-bladder and contract the sphincter. Doyon,[4] in 1894, drew the conclusion that excitation of the vagus nerve provoked dilatation of the sphincter mechanism surrounding the lower end of the common duct parallel to the contraction of the gall-bladder. Rost[12] later concurred in this opinion. Then, in 1917, Meltzer[10] pointed out that during storage of bile, the sphincter is contracted and the gall-bladder relaxed, and when bile is entering the duodenum, the gall-bladder is contracted and the sphincter relaxed. Westphal,[13] in 1923, after experimenting with guinea-pigs, concluded that a weak vagus stimulus resulted in a contraction of the gall-bladder and a relaxation of the sphincter of Oddi. Splanchnic stimulation caused relaxation of the gall-bladder and ampulla with contraction of the sphincter.

The mechanism of pain in extrahepatic biliary tract disease is not yet clearly understood, but increased tension associated with inflammation, spasm or stones would probably explain most cases. Biliary distress or colic in the individual whose gall-bladder has not been removed could be caused by increased pressure from occlusion of the cystic duct by stone, spasm, inflammation, stricture, band or tumor, or it could be caused by the same pathologic states along the common duct, principally in the region of the ampulla or sphincter. When a foreign body is passing through a narrow area, such as a sphincter, the pain may be due to the intrabladder or intraductal tension, or to direct irritation. Similarly, inflammation may in itself be intensely painful, but the resultant increased tension is doubtless just as important. In many attempts to relieve biliary tract distress, the release of tension with the concomitant lowering of intragall-bladder or intraductal pressure may be the all important factor.

The effect of drugs on the musculature of the gall-bladder has been extensively studied and an excellent review has been

presented by Ivy.[7] Their successful application in clinical gall-bladder disease is another problem. The effect of drugs on the lower end of the common duct is a more complicated study because we probably are dealing not only with a sphincter muscle entity, but also with a duodenal musculature which could, by its peristaltic action or state of tonus, affect the emptying of the choledochus. It seems, then, that the only means by which we can accumulate information as to the value of various drugs in relaxing extrahepatic biliary musculature and releasing intragall-bladder and intraductal tension, is the trial and error method, in patients with T-tubes or catheters remaining in the gall-bladder or common duct. By injecting opaque substances through the tubes, the relation of pain or distress to the distention and to the spasm or emptying may be observed in cholangiographic studies. Pressure within the gall-bladder and duct may also be measured, this latter method dating back as one of the early experiments in biliary tract investigations.

It was our observation in making delayed cholangiograms that injection of the opaque medium through T-tubes or catheters in some instances caused distress in the gall-bladder region, definitely referred at times to the back or right shoulder. In such cases, the roentgenograms usually showed the sphincter of Oddi to be contracted. Attempts to relieve the discomfort or to relax the sphincter by means of morphine were usually unsuccessful. If the intraductal pressure were continued, the pain would often eventually disappear even though repeated cholangiograms showed that the choledochal sphincter was still spastic. This was thought to be due to a depressing effect along the pain pathways rather than to any change of a mechanical or pathologic nature.

Atropine was tried on a number of these patients, but only occasionally did it alleviate the distress or cause the sphincter to relax. The combination of morphine and atropine was then tried, and again there was relief only at times. Epinephrine, 1 c.c. of a 1:1000 solution hypodermically, and ephedrine ¾ grain hypodermically, even when repeated in thirty minutes, were both ineffectual. Many times magnesium sulphate and fats, such as cream or olive oil, administered through a duodenal tube or directly by mouth, were beneficial, for the radiopaque solution could be seen passing freely from the common duct into the duodenum.

It was at this point in our investigation that one of our patients, as mentioned above, suggested the use of glyceryl trinitrate. We were fortunate enough to have several well demonstrated cases of spastic biliary dyssynergia in the hospital a few weeks later and immediate investigations with this drug were begun. Much to our delight, we proved cholangiographically that the opaque medium passed from the common duct into the duodenum within ten minutes after $\frac{1}{100}$ grain of glyceryl trinitrate was placed under the tongue. If the opaque solution was injected under considerable pressure and the in-dwelling catheter or T-tube clamped, any associated distress was immediately relieved. The use of this drug was then extended to patients whose gall-bladders had been removed some years previously and who were having recurrent attacks of biliary distress. Some of them were immediately relieved, although other antispasmodics had not been of much avail. Further utilization, in cases where remaining stones were found by making delayed cholangiographic studies, permitted us to observe the release of small stones from the common duct and to note that no relief was obtained if the stones were too large to get through the choledochal sphincter or if they were caught in a narrowed portion of the common duct. We believe this explains some of the failures to relieve post-cholecystectomy syndromes.

Glyceryl trinitrate was then employed in ordinary biliary colic where the gall-bladder had not been removed. Here again there was definite relief in some instances and failure in others. It could be assumed

that where relief was evidenced, spasm of of the sphincter of Oddi or some small stone or plug of inspissated bile caught within the sphincter had caused distention of the extrahepatic biliary tract including the gall-bladder, and that the glyceryl trinitrate had released the pressure by permitting free egress of bile into the duodenum. If relief was not obtained, it could be assumed that the common duct pathology was too extensive, or that the gall-bladder was distended by obstruction within the cystic duct, either by stone, inflammation or spasm of the debated collum-cysticus, and therefore the glyceryl trinitrate did not relieve the intragall-bladder pressure. In fact, this drug may affect the musculature of the gall-bladder and that of the sphincter in an entirely different manner, because of the complex reciprocal innervation pattern. It might be compared to atropine which, although it causes varying degrees of relaxation of the gall-bladder, also causes it to fill better by increasing the tone of the choledochal sphincter, according to Grebe.[5] If this latter be true, it is reasonably clear why atropine fails many times while glyceryl trinitrate is effective. On the other hand, we have seen glyceryl trinitrate afford no relief in some cases which did respond to atropine.

In an individual deprived of his gall-bladder, glyceryl trinitrate is, in our experience, the first choice of drugs. If the patient still possesses his gall-bladder, the drugs to be used can only be determined by trial and error. At present, we are attempting to determine the effect of glyceryl trinitrate on the intact human gall-bladder.

We have known for many years that morphine would not give relief in all attacks of biliary colic, or at least the relief would be delayed or incomplete. Some of our early cholangiographic studies revealed spasm of the lower end of the common duct which was not affected by morphine, and at times the accompanying distress was not satisfactorily relieved until pressure in the indwelling catheter or T-tube had been released. Other experiments have demonstrated that morphine produces an increase in the pressure within the common bile duct. This probably can be best explained by the fact that morphine stimulates the rhythmic contraction of the intestinal wall which in turn tends to serve as a secondary sphincter mechanism at the lower end of the common duct. Of course, there is usually partial to complete relief from pain because the morphine depresses the paths by which pain stimuli reach the consciousness. It is by removing the cause of the distress that we get quicker and more prolonged relief. If the distress is caused by a disturbance of the sphincter of Oddi, the combination of morphine and atropine may not give the desired relief, whereas glyceryl trinitrate may result in immediate cessation of pain. This may or may not be used in combination with morphine. The relaxing effect of glyceryl trinitrate on the sphincter seems to be greater than the spastic effect of the morphine on the duodenal wall. If it is a gall bladder-cystic duct affair, it appears that more satisfaction is obtained from the use of atropine, either alone or with morphine, but we have no undoubted proof that this will always prove the case, especially in the fact of active pathology. If it is not successful, glyceryl trinitrate is worthy of trial. Of course, there are those cases where associated spasm with increased pressure is of minor significance and antispasmodic drugs have no value.

Our greatest satisfaction in the use of glyceryl trinitrate has been in those cases where rather typical distress has recurred following removal of the gall-bladder. This may occur within the first few days after operation, or it may be delayed. In previous writings,[1,2,3,6] we have discussed and demonstrated the not infrequent presence of spastic biliary dyssynergia and how it may be associated with biliary distress. By cholangiographic studies, it has been proved that this spasm can be released by glyceryl trinitrate, and with its release, the associated distress disappears. During

the last eighteen months, all patients who complain of any post-operative distress which might be related to a biliary tract syndrome have been given glyceryl trinitrate tablets, 1/100 grain, to be placed under the tongue. They are repeated several times during the course of a few hours if necessary, or they may be used three times a day for a short period. They not only relieve the spasm and permit free drainage from the common duct into the duodenum, but inspissated bile or stones may find easy exit. If there is a catheter or T-tube in the common duct, it is irrigated once a day with normal saline, olive oil or lipoiodine, following the administration of glyceryl trinitrate. On several occasions, we have been able to remove residual stones from the common duct by following such a régime. When patients come to us now with symptoms persisting after cholecystectomy, and if operation is not definitely indicated, glyceryl trinitrate is prescribed in conjunction with cream, olive oil, or occasionally, magnesium sulphate. Cholagogues may be given at the same time in the hope of establishing a free flow of bile to wash out any remaining small stones or inspissated bile.

SUMMARY

This article concerns the management of some of the failures of biliary surgery, proved to be due to spasm of the sphincter of Oddi, and the use of the drug, glyceryl trinitrate, as a means of relieving this postoperative biliary distress. It is believed that an appreciation of the cause of some of the failures following biliary surgery may place these cases in the category of successful surgery. The use of glyceryl trinitrate and atropine in the management of biliary colic in non-operative cases is also discussed. It appears that glyceryl trinitrate is valuable in biliary colic due to sphincter of Oddi spasm while atropine is more apt to relieve spasm of the collum cysticus at the neck of the gall bladder.

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AMBULATORY METHOD OF TREATING FEMORAL SHAFT FRACTURES, UTILIZING FRACTURE TABLE FOR REDUCTION*

ROGER ANDERSON, M.D., F.A.C.S.

Orthopedic Surgeon, King County Hospital

SEATTLE, WASHINGTON

SUCCESSFUL reduction of femoral shaft fractures can be more quickly and efficiently achieved when direct control of the recalcitrant upper fragment is obtained, while for early and complete restoration of function, it is imperative to perfect an immediate reduction sustained by positive immobilization of each fragment. Such approximation and fixation in all types of fractures of the shaft of the femur can be accomplished when a specialized form of skeletal countertraction is employed.

FIG. 1. Comminuted fracture of the middle third. Demonstrates advantages of this physiologic method of immobilization.

Transfixion of the upper femoral fragment may not, at first thought, seem feasible. But, since the sole means of

* Read before British Columbia Medical Society, Vancouver, British Columbia, September, 1935.

proximal insertion is a pair of short half-pins put into the lateral aspect of the superficially placed greater trochanter, it incorporated in plaster, thereby actually nailing the fragments to a short cast, extending only from hip to knee.

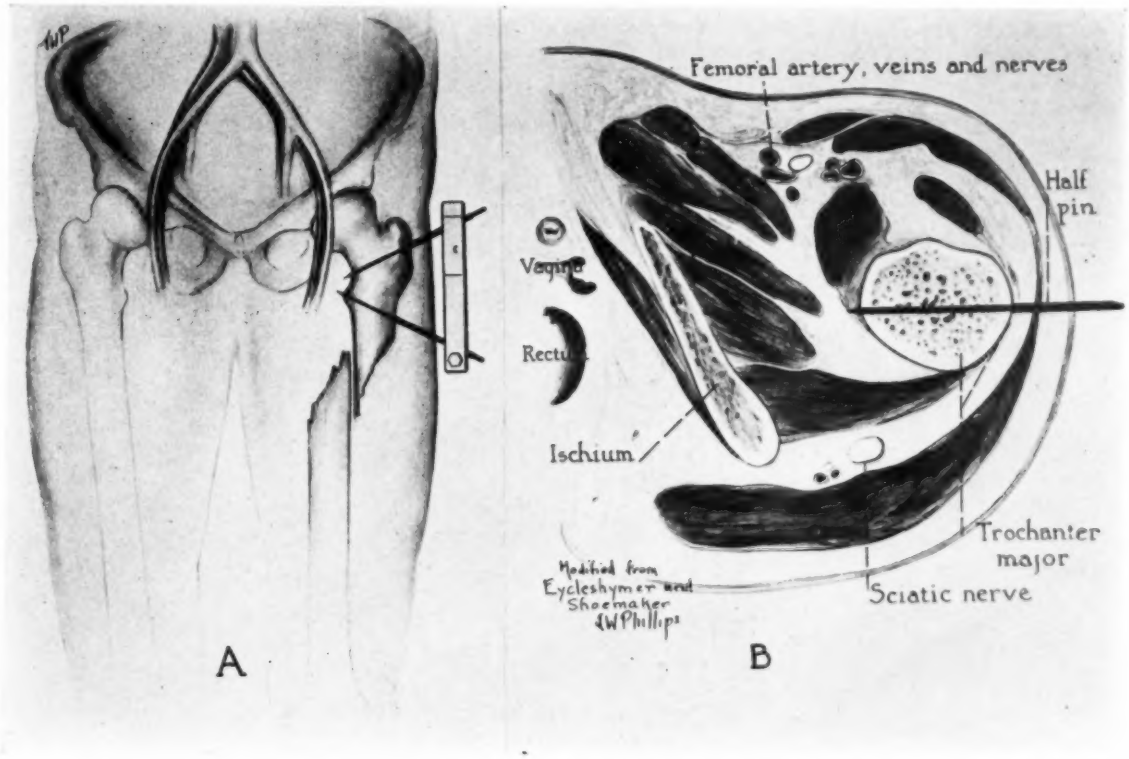


Fig. 2. Diagrammatic sketch showing anatomic practicality of transfixing upper fragment. A, shortening alone would not result in apposition of parts. This spiral fracture is referred to as a locked fracture. See text for technique of reduction. B, cross section of left upper thigh of female at the level of the lesser trochanter, demonstrating relationship between half-pins and femoral vessels.

is apparent that the procedure is practical as well as safe. (Fig. 2A.) For anatomic reasons, these half-pins, as the name implies, transfix but half of the thigh, although they completely transfix the upper femoral shaft. (Fig. 2B.) As the half-pins are inserted into the superior fragment at an angle to each other, with their protruding ends connected to an adjoining bar, the fragment can consequently be manipulated as if grasped with forceps.

Technical Advantages. Transfixion of the superior fragment with half-pins and dual transfixion of the distal fragment by Steinmann pins or Kirschner wires serve a triple function: (1) they supply skeletal traction and skeletal countertraction; (2) they furnish the means for direct manipulation of each fragment; and (3) they provide absolute immobilization when

Immediate movements at knee and hip, without disturbing apposition, are made possible by incorporating both sets of transfixions in the plaster cast. (Fig. 1.) With complete immobilization so attained, there can obviously be no loss of reduction. With minimum encasement of the thigh, which immobilizes neither hip nor knee joint, benefits from ambulation accrue. It is evident that on drying of the plaster, immediate crutch ambulation is provided for. Patients may be fully dressed throughout convalescence, and the latter is practically pain-free. There is, therefore, added economy in a few days' hospital stay.

Traction force for reduction may be obtained through various agencies: the fracture table; the anatomic femur splint;[1] by means of weights and pulley; or by manual traction.

Fracture Table Reduction Technique. After a preliminary hypodermic and skiagrams in two views, the patient is placed on the fracture table with the feet securely bandaged to the foot plates, and after some traction has been exerted and the table top dropped, the upper thigh and the knee are surgically prepared for the transfixions.

Superior transfixion is demonstrated in Figure 3. Because the lower half-pin penetrates the dense cortical bone of the shaft, it is best to employ a carpenter's drill or an electric motor drill. When *x*-rays are taken to check position and depth of insertion of the half-pin, this assembly should be covered with a sterile towel. A good rule is to have the ends of the half-pins pass well beyond the medial cortex of the bone; therefore, should the radiographs demonstrate that penetration is inadequate, the half-pins require no preparation for deeper insertion.

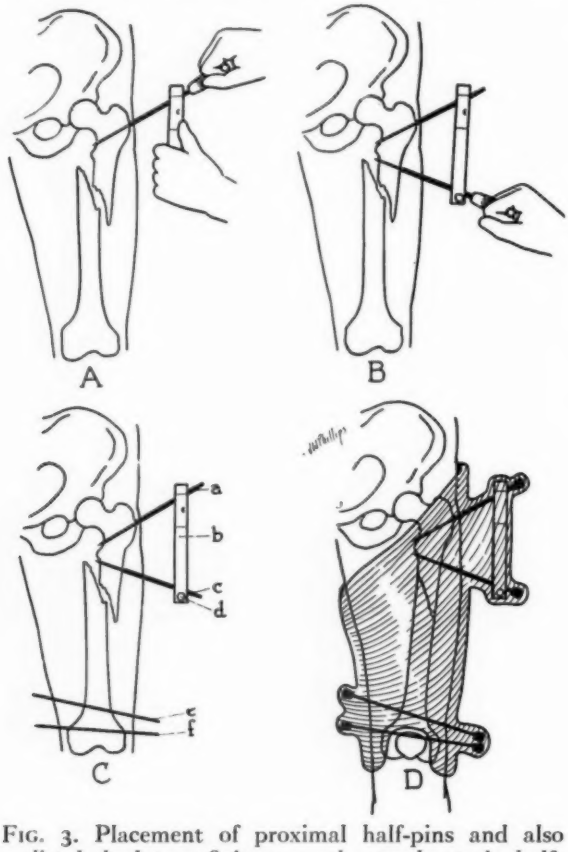


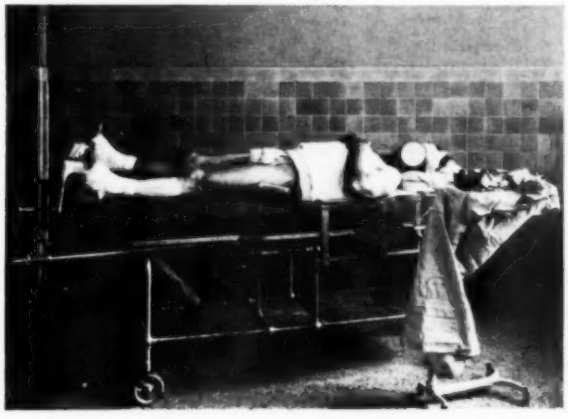
FIG. 3. Placement of proximal half-pins and also distal dual transfixions. A, the trochanteric half-pin, *a*, is drilled by the right hand into the center of the greater trochanter, while the left hand holds the clamping bar, *b*, parallel to the thigh. B, the locking half-pin, *c*, after passing through the oblique hole in the clamping bar, is drilled completely through the shaft and locked in position by tightening the nut, *d*. The half-pin units are made in three sizes: large, medium and child. C, the most distal transfixion, *f*, either pin or wire, or a pair of half-pins if preferred, is inserted at the superior edge of condyles while the other transfixion, *e*, is passed through at a point about 2 inches superior to the condyles. D, after reduction the transfixions are securely incorporated in the short cast, which extends only from the hip to the knee joint.

Two distal transfixion pins or wires are routinely employed, in order that the knee

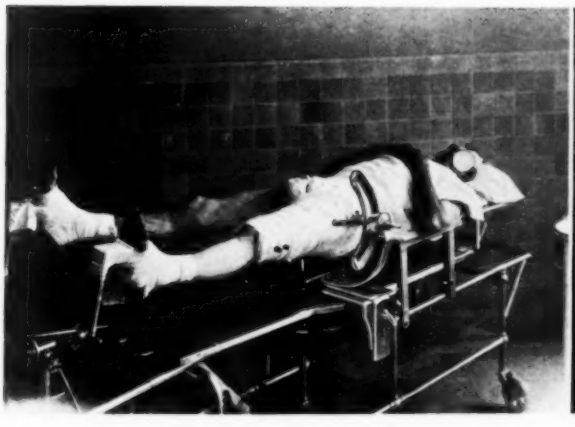
joint be left mobile. If, however, only one distal transfixion is desired, the cast must of necessity be extended down to the level of the mid-calf, and the distal transfixion should be put in at an oblique angle to the transverse axis of the knee joint. Such obliquity provides better immobilization and prevents any sidewise sliding of the distal fragment on the transfixion. While awaiting Roentgen findings on the location of the half-pins, the most distal transfixion is inserted into the superior edge of the lateral condyle (Fig. 3c), the uppermost distal pin or wire being placed about two inches above the condyle at an angle so that the *dual distal transfixions are not parallel* to each other.

Correction of the lower fragment is now made by adjusting the traction nut, and by rotating the foot plate of the fracture table. Manipulation of the upper distal transfixion, either manually or by tying this pin with bandage to the overhead bar of the fracture table, corrects anteroposterior displacements.

The upper fragment may be manually manipulated through the "handle," as it were, of the half-pin unit, its position held by the assistant while plaster is applied. However, a more reliable procedure is to



A



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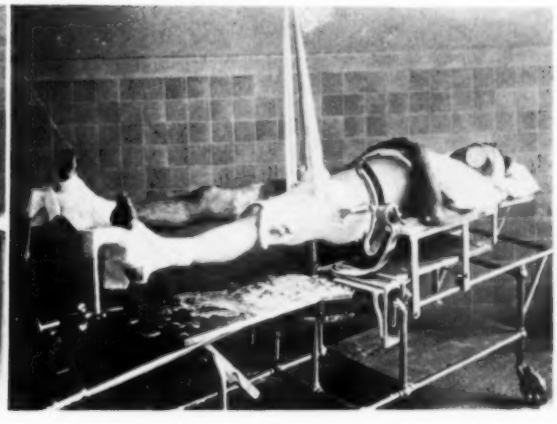


C

Fig. 4. Demonstration of steps in reduction on a Hawley fracture table. (See also Fig. 6.) A, first stage, the placement of half-pins in the superior fragment and two transfixions, or half-pins if desired, into distal fragment. B, second stage, the attachment of the mechanical hand or clamp to the half-pins, the clamp being universally adjustable and operable equally well for child or large adult, as well as for either leg. Here a Hawley table is used, but the clamp may be fastened to any standard type of orthopedic table. If clamp is not available, a reliable, steady assistant can manipulate and manually hold the half-pin unit while the plaster is applied. C, third stage, the application of a padded or unpadded cast is not made until a perfect reduction with direct end-to-end contact, radiographically proved, has been achieved. All transfixions must be securely incorporated in the cast.

use the fracture table mechanical arm. (Fig. 4.) This bracket clamp, devised to be quickly fastened to any regulation fracture table, may be adapted to the age and the size of the patient, and regulated for either the right or the left femur. (Fig. 5.) The clamp or so-called "mechanical arm" is so constructed that all manipulations, mechanically performed, are made on the normal anatomic axis with the head of the femur as the center. Reduction should be radiographically checked until apposition is practically perfect.

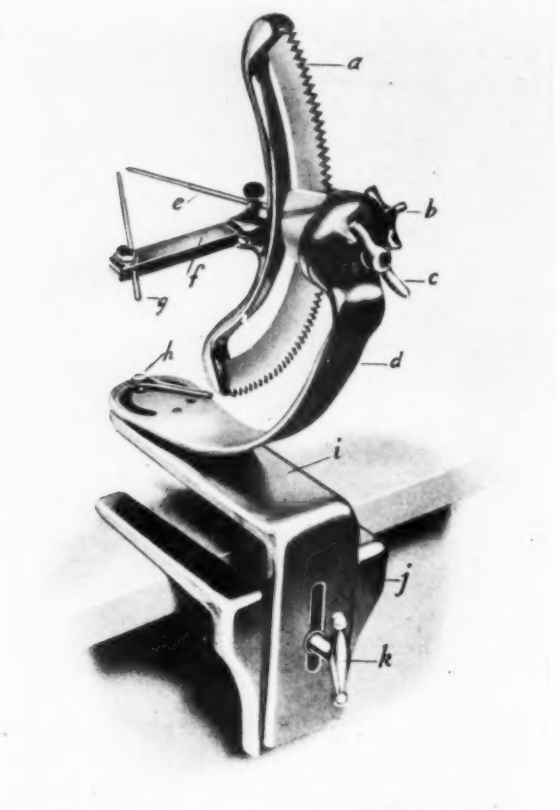
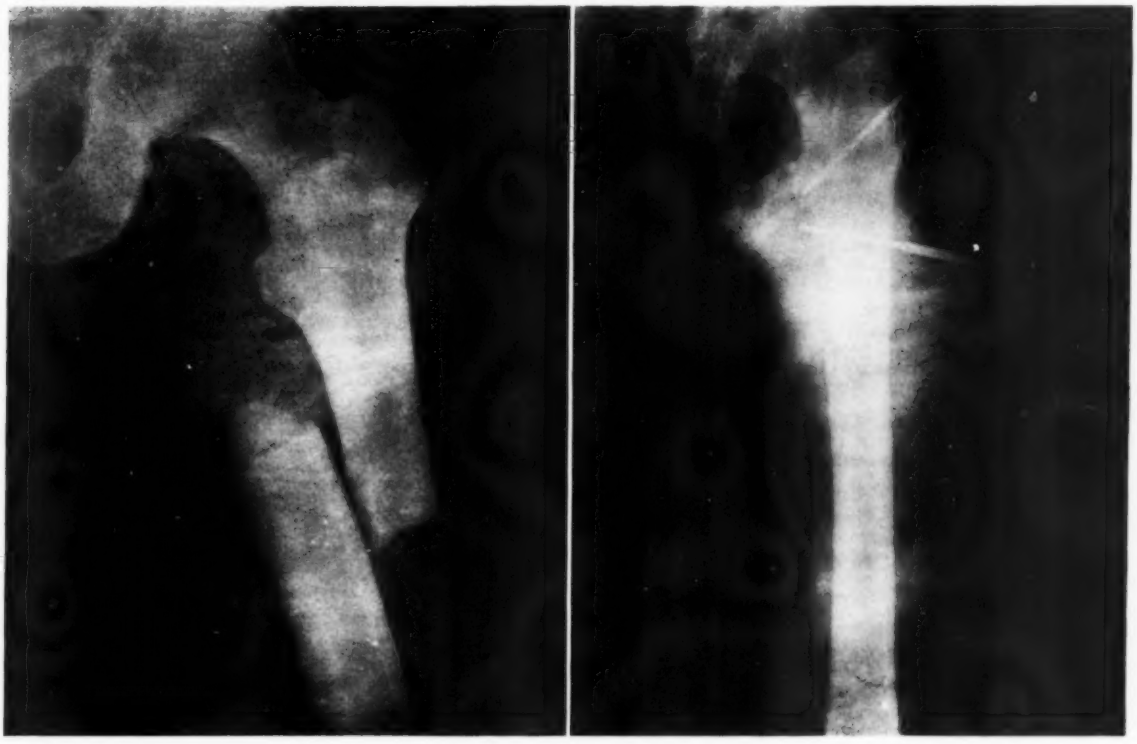


FIG. 5. The universal fracture table clamp or mechanical arm is constructed so that it can be quickly fastened to any of the standard fracture tables by tightening handle, *k*. The universal bracket, *d*, is pivoted under the hip, and abduction and adduction corrections made by swinging and locking this bracket at *b*. The rotating arc, *a*, through its hand-operated handle, *b*, rotates the fragment into position, and as the arc is swiveled and locked at *c*, all flexion and extension deformities can be corrected. The half-pin assembly, *f*, is attached by tightening its nut. Construction is such that rotation and all manipulations, regardless of the size of the patient, are anatomically made with the center of the hip as the axis.

Immobilization is obtained through a short cast, either padded or unpadded, which extends only from the crest of the ilium to the knee, and *securely incorporates the transfixions*, the pin-ends being covered with corks and plaster. (Figs. 1 and 18.) Directly upon the setting of the plaster, the patient is removed from the fracture table.

After-Care. This is minimal. Only occasionally is it necessary to split the cast; however the plaster over the groin, and back of the knee is cut away to provide mobility for the hip and knee joints.



A B

FIG. 6. Roentgenogram of fractured femur in same patient shown in Figure 4. A, before reduction. B, after reduction.

FIG. 7. Same patient as shown in Figure 6. Final result thirteen and one-half months after reduction. Bowing caused by unprotected weight-bearing following removal of the transfixions.

Transfixion wounds need no care until the transfixions are removed, usually eight to twelve weeks later, depending on callus formation.

hospital. At first it is best to use crutches, but in the late convalescence a cane usually suffices. Ambulation apprehension soon disappears and convalescence should not

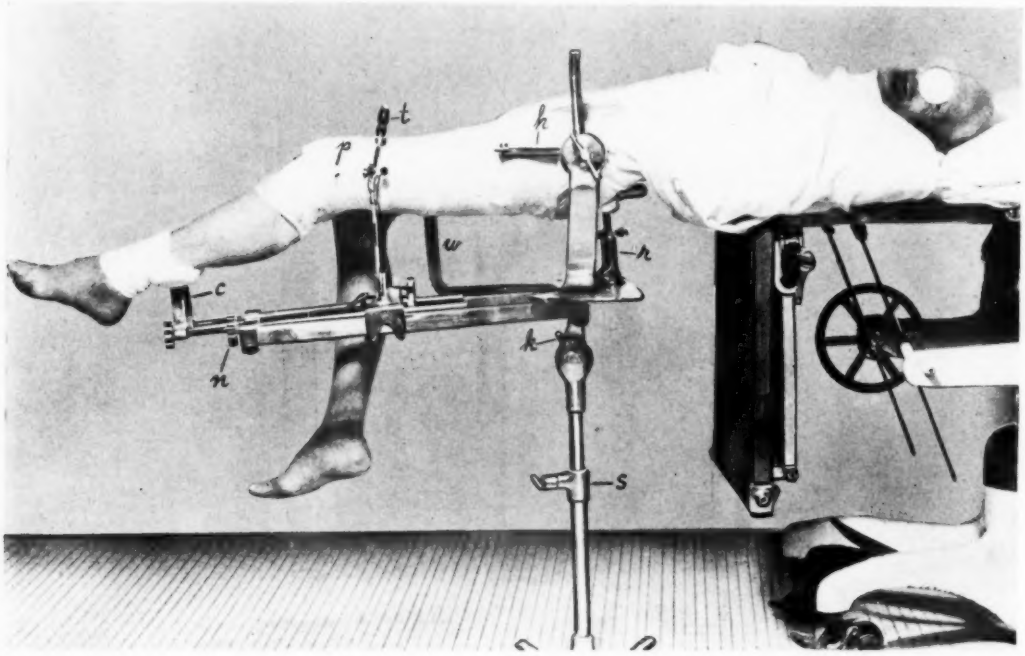


Fig. 8. Comminuted fracture of middle third, reduced under spinal anesthesia. Here the mechanical arm is pivotly fastened to the upper end of this anatomic femur splint, and again all mechanical manipulations are made with the head of the femur as the center. The sacral rest, *r*, an integral part of the apparatus, is adjustable to fit any sized patient.

The uppermost of the two distal transfixions (in this case a Kirschner wire in the Tautner *t*) is fastened into the horseshoe, which is pivoted for lateral or medial adjustments; through a gear and rack arrangement, this distal fragment also is rotated on the normal anatomic axis. Furthermore, by pressure down or up on the knee joint, anteroposterior displacements are overcome. Traction supplied by turning nut, *n*, of the traction rod. When available, we prefer to use the anatomic femur splint, as the correction is more easily obtained, while through mechanical locks, apposition is positively held during the application of the plaster. Furthermore, the construction of the splint and its adjustable stand, *s*, was devised to permit free and easy access for fluoroscopic and *x*-ray examination.

Skiagrams are repeated several times. Separation of fragments, caused by overtraction at the time of reduction, unequal contraction in the drying of the plaster, or later slight absorption at the ends of the fragments, as a result of circulatory trauma, calls for correction in order to avoid delayed union. Such a condition is quickly remedied by removing a thin, transverse section of the cast.

Ambulation is permitted as soon as the patient is out of shock and the plaster dried, and within a few days the patient, fully dressed, can generally leave the

cause much discomfort. Pain, if present, is usually due to faulty technique. However, the continued pull of the skin against the transfixions in moving about may result after a few weeks in some distress, accompanied by a purulent discharge. As this secretion is sterile, however, no attention is necessary, and dressings are left undisturbed for the usual time limit. Should these complications become aggravated, it is advisable to keep the patient in bed from a few days to a week. Another possible source of discomfort and discharge may be bony absorption around

the ends of the half-pins, a result of failure to transfix the femur completely with the half-pins. Here again, merely keeping the patient off his feet for a few days usually suffices to allay the difficulty, and only occasionally is it necessary to reinsert the half-pins.

Suppuration and distress around the distal transfixions from sidewise movements may occur on recession of swelling in those cases in which the two distal transfixions have been inserted parallel to each other. For this reason these transfixions should be placed in oblique relationship to each other. Danger of infection is practically nil; we have yet to have our first case of bone infection from the use of these transfixions. An itching of the skin may cause an uncoöperative patient to scratch and infect the pin wounds, but this danger can be avoided in many instances by using larger dressings and covering their vulnerable aspect with transverse strappings of adhesive. With weak-minded or senile patients, it may be advisable to extend the cast upwards in the form of a spica, with plaster so thin over the abdomen that the body portion is semi-flexible.

Fig. 9. Comminuted intercondylar T-fracture into knee joints before reduction. Since the distal portion was so short, a single Kirschner wire was put through both fragments of the T, while the second transfixion, a Steinmann pin, was put through the upper tibia, with cast 6 inches below the knee. Latter transfixion removed at an early date.

Locked Fractures. The fracturing force that causes a spiral fracture will frequently displace the fragments so that the fractured surfaces do not face each other. As illustrated in Figure 2A, traction may overcome all shortening in this type without effecting apposition, and therefore to get reduction by traction, it is actually obligatory to lengthen the thigh from one to several inches before the fragments slip past each other. We refer to these displacements as the locked fracture. Such cases in the past

have come to open operation on the diagnosis of intervening muscle.

By mechanical manipulations of the half-pins, these fractures can now be simply unlocked by passing an end of one fragment either above or below the other fragment so that both fractured surfaces face each other. This is an initial maneuver, so that when traction is applied, reduction is quickly achieved.

Fig. 10. Same patient as in Figure 9, after reduction.

Although radiographs may reveal just a simple spiral fracture, subsequent careless handling in moving patient to operating room may result in a slipping with consequent locking of the fragments. If traction fails to effect speedy reduction, the fracture should be checked by fluoroscopic or *x*-ray examination; and if locked, traction is released, and the fragment unlocked, after which traction soon results in reduction.

In subtrochanteric fractures, abduction, flexion and external rotation of the short upper fragment can be readily corrected by routine measures. However, the upper half-pin should be inserted into the superior edge of the trochanter, although the occasion may require the smaller half-pin unit with half-pins in closer proximity.

In supracondylar fractures, posterior displacement of the distal fragment is overcome by pulling anteriorly on the uppermost distal transfixion until the knee joint is fully extended, with the lower femoral fragment in a straight line with the tibia. After this the fracture is routinely reduced. This procedure is more easily accomplished by utilizing the overhead suspension rod set into the center post of the fracture table.

Comminuted fractures are managed routinely, except that plaster should be more snugly applied. The infrequent case in which a large, loose central fragment is displaced (usually to the medial side by

the pull of the adductor muscles) can be controlled as follows: A large hole over the displacement, or in fact the quarter section of the side of the cast, may be cut out in order to force against the displaced fragment a roll of sheet wadding, three to four inches in diameter and about six inches long, so that it contacts the major fragments when a bandage is passed over the roll and around the remaining part of the cast.

The wounds of compound fractures may be debrided and primarily closed, or packed with Orr's vaseline gauze. If the anatomic splint is employed, the wounds may be chemically lavaged, or treated with maggots or any other accepted method that the surgeon elects, because the thigh may be left fully exposed, reduced and completely immobilized in the splint for as many weeks as desired.

End Results. The technique described has been routinely used on our service for all femoral shaft fractures, regardless of location or type. Reduction has been obtained in every case; we have not as yet had a single incidence of a transfixion osteomyelitis; we have obtained initial bony union in every case when a period of four months has elapsed since date of accident.

Fig. 11. Final results in patient shown in Figures 9 and 10, three months later.

PRECAUTIONS

We are fully cognizant of the fact that a surgeon is not made by the mere purchase of a scalpel, but we do know that the mechanically inclined surgeon can obtain

A B C D

FIG. 12. Fracture of distal third, reduction by D. M. Meekison, orthopedist, of Vancouver, Canada. In a personal communication, Dr. Meekison states that since his acquaintance with this method, he has employed it routinely on all femoral shaft fractures, obtaining reduction in all cases, and bony union in every case of over three months' standing.

A B

FIG. 13. Patient aged 9 years with comminuted fracture of both femora. A, before reduction. B, after reduction.

excellent fracture results if he adheres closely to the technical details of this new method and avoids certain pitfalls. We therefore admonish the surgeon:

Fig. 14. Same patient. Final result a little over three months later.

To locate fragments by probing with Kirschner wire.

To pierce through and beyond the medial cortex with the medial half-pins.

To unlock fracture before attempting reduction.

To obtain direct end-to-end contact reduction.

To verify apposition by *x*-ray films before applying plaster.

To avoid excessive traction with fragment separation.

To employ the best grade of plaster bandages.

To incorporate transfixions securely in the cast.

To repeat Roentgen examination during convalescence.

To instruct the patient to keep fingers and sticks out of the cast.

Fig. 15. Same patient as in Figures 13 and 14. A single distal transfixion was used on each side and casts extended to the lower leg. since knee stiffness is not a factor in children. A few turns of plaster bandage may be put around the waist in children or in senile patients, in order to keep their hands away from the half-pins.

To refrain from any inspection or dressing of pin wounds.

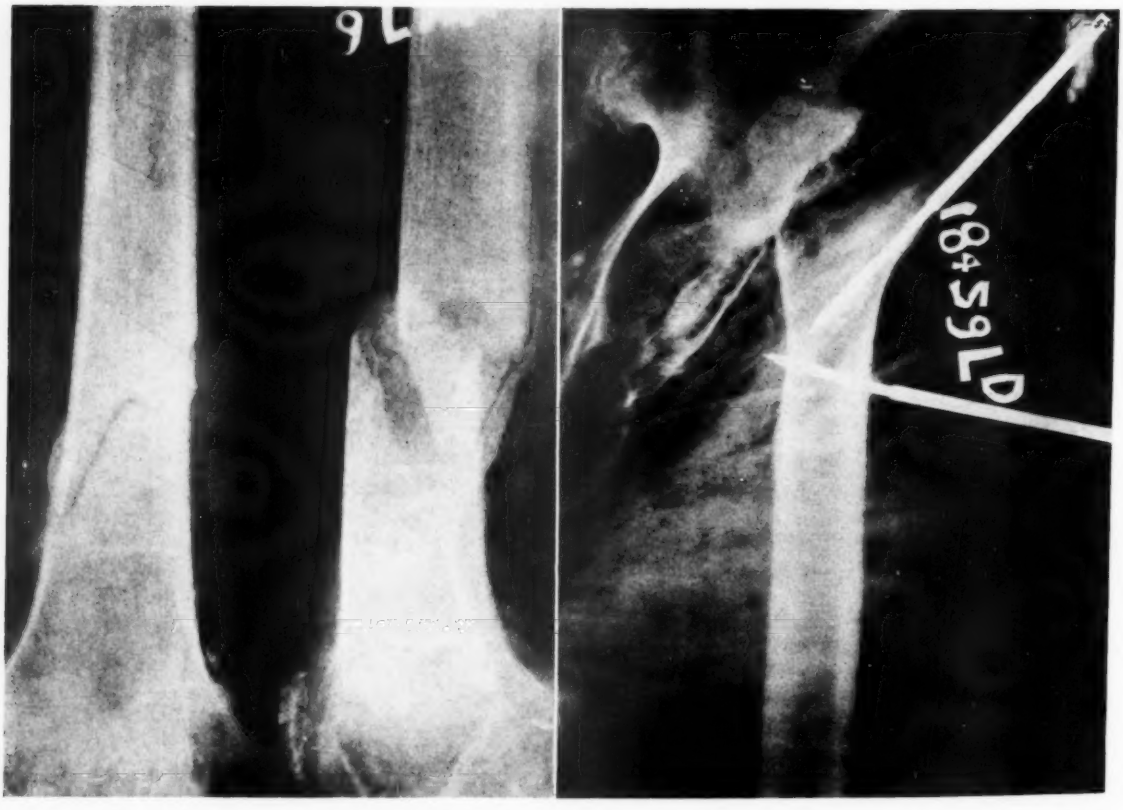
To leave transfixions until *x*-ray callus is revealed.

To protect green callus following removal of transfixions.

SUMMARY

For femoral shaft fractures a simple means of transfixing the proximal fragment provides a method for immediate, successful reduction. Furthermore, this system with its plaster incorporated skeletal traction and countertraction transfixions supplies absolute immobilization,

Fig. 16. Pathologic fracture, tabetic, distal third.



A B C

Fig. 17. A and B, final result in case shown in Figure 16, three months later. C, reduction and bony union regardless of the fact that fifteen years previously patient had received fracture of neck with non-union, complete absorption of the head and resulting dislocation of the trochanters upward.

despite the fact that only a short plaster cast is applied to the thigh. The physiologic and economical advantages are numerous, including immediate freedom of hip and knee joint movements, a brief hospital stay, minimum after-care and painless convalescence, benefits epitomized by a fully dressed patient, completely crutch-ambulatory.

Fig. 18. Same patient as in Figures 16 and 17. Patient was crutch-ambulatory on the ninth day, in spite of the absorption of the femoral head and the tabetic condition, plus the recent spiral shaft fracture.

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THE END RESULTS OF EARLY AND DELAYED TENDON SUTURE

C. R. G. FORRESTER, M.D.

CHICAGO, ILLINOIS

THIS paper is not written with the idea of invading the field of the reconstruction surgeon. It is for the purpose of showing the surgeon who has an emergency on his hands and who cannot reach a specialist, what can be done and what our methods are, and the end results.

From an industrial standpoint, the ability to cope successfully with this type of injury at the time the injury occurs means not only a saving to the company involved, but also a good working member for the patient. It also avoids a long, protracted disability, a doubling up of surgery, which even in the best of experienced hands, does not always result in a functional member.

The writer has been specializing in this work for some thirty-three years, during which time he was forced to work out some technique that would be successful and of benefit to all concerned. The results have not always been 100 per cent perfect at my hands, for, under the best of care, complications will develop, though in the majority of cases the results have been gratifying.

I wish here to emphasize the fact that in handling this type of injury the surgeon should look upon it as much of a major operation as any performed on the human body. To say the least, it must be just as delicately carried out. Avoid traumatizing the tissues as much as possible, as they have already been traumatized enough. Restore the continuity of the tendon and its sheath as carefully as possible.

Preparation. This work usually should be conducted under general anesthesia unless one is thoroughly trained in the use of local anesthesia where the blocking can be successfully done *above the field of operation.*

The field should be shaved and carefully scrubbed with soap and water, drenched with sterile water, then alcohol, then ether. Field towels and covers should be carefully placed. The scrubbing and preparation of the operator must be as thoroughly carried out as for any major operation.

The debriding of the surface tissues must be most thorough, no matter how long it takes; the surface must have a *clean field before approaching the tendon.*

Do not try to repair the tendon through a small approach wound; enlarge the wound sufficiently to expose the field comfortably. You are endangering the situation in working through a small wound; infection can occur through a small traumatized wound just as quickly as through a large one. In enlarging the wound, study the field, so that your surgical incision, when healed, does not interfere with ultimate function.

With the tendon exposed, a careful scrutiny will give some idea of procedure. It is necessary to use the smallest of artery forceps, scissors, and knives to avoid trauma. *A tendon should be handled just as carefully as a nerve.* The sheath and tendon should be very carefully debrided of all dirt. I have even dissolved grease with small cotton applications of gasoline, followed by alcohol and then by ether. When one is satisfied that everything is as clean as possible, the surgical repair can be considered.

The only retention suture for the tendon is silk, as it must not only last longer than catgut, but stand spasmodic contractions of the muscles attached to the tendon. The silk suture must be applied in such a manner that knots are buried within the tendon substance. Figures 1, 2, and 3 show the

author's method of placing the tension sutures and burying them. If this is not done, any exposed knots of silk will cause irritation and subsequent sinus, so that ultimately the silk will have to be removed. Following this, the tendon sheath must be very delicately closed with interrupted catgut sutures at close intervals as shown in drawing, to keep the silk knots from escaping, also this catgut closure helps very much to prevent adhesions.

The size of silk used should be equivalent to a Number 1 chromic and it should be *braided silk*. The catgut by preference should be double Number 0 Pyoktannin. Therefore, the smallest of instruments, such as needles, forceps, etc., must be used.

After this repair is completed, any satisfactory antiseptic may be used before closing. The author uses one drachm of powdered iodoform to two ounces of ether.

The superficial tissue closure should be carefully studied and the smallest of catgut Pyoktannin used; the skin is closed *without tension* with fine black silk.

A large fluff dry dressing with the member in a position of rest which produces the least tension on the entire limb is absolutely necessary.

The tendon repair technique is illustrated in Figures 1, 2 and 3.

Case 1. (Fig. 5.) This case illustrates the effect of a comparatively minor injury with results following immediate surgery.

Date of injury, January 31, 1935 at 2:30 P. M.

Date of first treatment, January 31, 1935.

History. Fell to the floor with electric light bulb in his hand; bulb broke.

Examination. One-third inch transverse laceration in middle of palm of right hand.

Findings. Inability to flex middle finger; numbness of middle finger.

Operation. Under 2 per cent procaine regional block at wrist. Findings. Both flexor sublimis and profundus completely severed, also median sensory nerve branches to both sides of same finger.

Surgical Repair. Suture of tendons and nerves.

Progress. Dressing. Flexion of wrist and fingers to take tension off tendons and nerves. 1500 units tetanus.

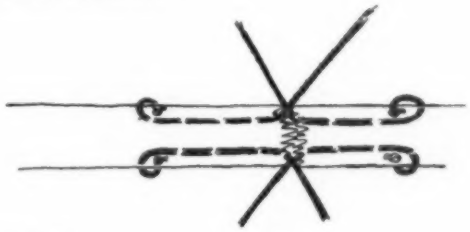


FIGURE I
TENDON SUTURE REVISED
BRAIDED SILK DRAWN
THROUGH TENDON-

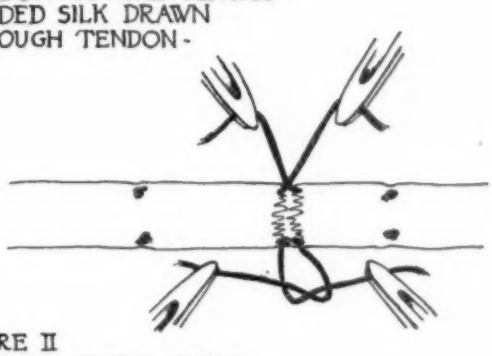


FIGURE II
SURGEON TYING FIRST
KNOT WHILE ASSISTANT
MAINTAINS APPROXIMA-
TION OF TENDON-

FIGURE III
TENDON SHEATH
SUTURED WITH
CATGUT-

Figs. 1, 2, and 3.

Left hospital day following operation with prognosis of post-operative recovery of motion in about two and one-half months and sensation in four to six months. Sutures removed on thirteenth day; slight voluntary motion. Gradual exercises started February 20, 1935. Touch and pain returning on March 2, 1935. Function of finger, half flexion.

March 27, 1935, slight numbness still present over median distribution, only one-third limitation in flexion of finger. Returned to work April 1, 1935. On April 1, 1935, flexion of finger only one-fourth limited. May 3, 1935, flexion of finger limited between 10 and 15 per cent,

sensation improving. Figure 5C shows amount of function upon discharge.

Case II. Showing results of immediate repair. (Fig. 6.)

fingers; atrophy of muscle between thumb and first finger; some atrophy of the dorsal surface of the hand. Full flexion and extension of the hand on the wrist. Slight contracture of the ring

A B C

Fig. 4. A, longitudinal incision (January 12, 1937). B, lateral view of flexion of fingers from ulnar side (January 12, 1937). C, full flex of fingers from radial side (January 12, 1937).

Date of injury May 18, 1934.

History. While cleaning top of telephone booth with glass roof, lost balance and fell through roof, cutting right forearm.

Examination. A three-inch crescentic laceration some two inches above wrist transversely. Removed to Chicago Memorial Hospital and repair made under general anesthesia, taking three hours' time.

Findings. Ulnar nerve severed; dorsal cutaneous nerve severed; median nerve not involved; ulnar artery severed.

Operation. Ulnar nerve sutured and four flexor tendons on ulnar side sutured, using the writer's technique. Hand dressed with dry dressing in position of flexion.

Progress. Discharged from Hospital May 24, 1934. May 26, 1934, sutures removed. Instructions given as to preliminary exercises of the digits. Gradual active and passive motion. June 14, 1934, good motion in flexion and extension of all fingers and thumb. Numbness in little finger and ulnar side of ring finger. Still in supportive splint. June 27, 1934, good grip in right hand. Good spread of fingers with slight restriction between middle and ring finger but improving. November 8, 1934, general interosseous atrophy improving, good grip, full motion of the hand on the wrist. November 17, 1934, a specific loss of the use of the hand of about 15 per cent. No necessity for further treatment. (This man worked right along.) Figure 6 shows the ultimate result.

Case III. Demonstrating the difference between immediate repair and reconstructive surgery. This is a case where apparent repair was made, and which subsequently came under the attention of the author.

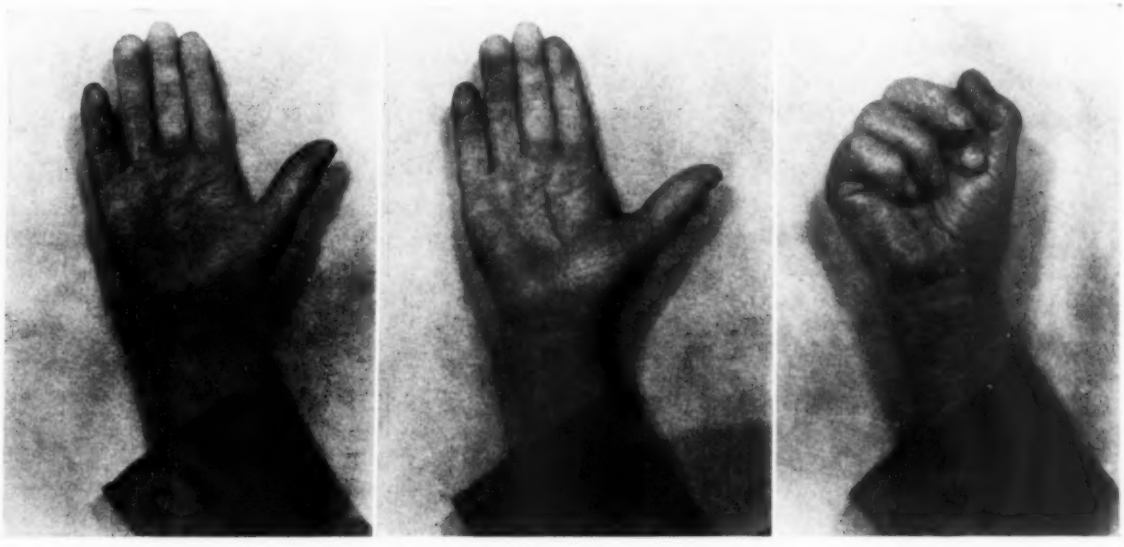
History. Carrying a bucketful of brass up an incline, stumbled and fell, lacerating right forearm just above wrist line. Supposed repair of tendons, nerves, etc.

Examination. May 10, 1934, by the author. Showed a ragged laceration on the volar surface of the right wrist extending from the ulnar lateral side to radial. Entire hand and fingers swollen. Complaint of numbness of all fingers, inability to differentiate between sharp and blunt test of sensation.

June 8, 1934, wounds healed without infection. Heat and massage to keep joints free pending ultimate determination of surgical

repair. Advised waiting thirty days before surgical intervention.

Operation. June 29, 1934. A longitudinal incision was made and structures exposed. It was found that the superficial flexors of the third and fourth fingers had been sutured to the proximal end of the ulnar nerve, the distal ends being free in the tissues. I had to dissect tendons away from the nerve and on account of complete severance of the tendons was not able to approximate flexors of second, third and fourth fingers to the distal structures, transplanted flexor carpi ulnaris into a conjoined fixation of third, fourth and fifth finger flexors. Sutured ulnar nerve. On dissecting the median nerve, found it sutured to the tendon of the first and second fingers flexor sublimis. This was changed and the tendons sutured to their proper structures. There was found an interstitial fibrosis of the median nerve; the nerve therefore had to be resected down to normal axis cylinders. I could not approximate the median nerve without flexion of elbow and wrist, but finally accomplished this. Following



A B C

Fig. 5. A, small laceration in palm of hand causing severance of superficial and deep flexors and nerves of second finger. B, line of incision for exposure of tendons and nerves. C, result four months later.

A B

Fig. 6. A, location above wrist with end result. Ability to adduct fingers. B, shows ability to flex fingers and thumb.

sutures of the nerves and tendons the member was placed in a cast with the elbow in mid-flexion and the wrist flexed as far as possible.

Period of time spent on this surgical repair was from 9:15 A.M. to 12:30 P.M.

Prognosis at this time is guarded. Temporary disability is estimated at five to six months, with the arm to remain in a cast at least forty-five days, with gradual stretching of the elbow and wrist to follow.

Progress. July 7, 1934, discharged from hospital. Window in cast. Sutures removed. Healing per primam. July 24, 1934, cast cut, sinusoidal treatment to stimulate muscles. A posterior cast is worn.

September 4, 1934, slight flexion and extension, motion voluntary in thumb and fingers. No splint worn. Beginning return of sensation of median and ulnar nerves with some evidence of tendon function. October 13, 1934, some of the silk sutures had to be removed because of irritation. October 26, 1934, continued improvement in flexion and extension of fingers, grip improved. November 24, 1934, Volkman splint applied to increase extension of fingers on wrist. Heat, massage and sinusoidal galvanism continued. December 11, 1934, excursion in the wrist increased; extension and flexion of the fingers improved. January 8, 1935, sensation returning along ulnar distribution areas, hypersensitive, also along median area. Ring finger somewhat contracted; index and little fingers can be fully extended. January 29, 1935, musculature of hand developing fair grip. March 6, 1935, still receiving massage, muscle reeducation and sinusoidal galvanism. Able to flex fingers to palm to within half an inch. March 30, 1935, slight limitation in extension of middle fingers. Nerves, both median and ulnar, hypersensitive, indicating regeneration. June 12, 1935, tissues of hand softer and grip stronger. Brings fingers to palm actively; fingers can be curled passively. Sensory function of median and ulnar nerves found to be normal to epicritic and protopathic tests.

June 17, 1935, patient returned to his regular duties. Examination on August 3 showed an increased flexion of fingers and good grip; contractural deformity of middle finger only. Specific loss according to Industrial Commission, was one-third of hand. The patient is working at his regular occupation and getting full pay.

CONCLUSION

These three cases show, first, the end results of immediate tendon surgery; second, the reduction in medical, surgical and hospital cost; third, the early return to work as compared with a later reconstruction, as shown in Case III. The procedure outlined is not one for the office, but for the hospital.

SOME THEORETICAL CONSIDERATIONS IN THE CONSTRUCTION OF ACTIVE SCOLIOSIS BRACES*

JOSEF WOLF, M.D.

DAVENPORT, IOWA

IN Schanz' textbook on orthopedic technique, published in 1923, not less than seventy-four different types of "active" scoliosis braces are described. Active braces in this connection mean orthopedic appliances, worn only or usually during the day, which try to correct the deformity through the use of pads, strips, cuffs, shells, etc., and are equipped with elastic forces (rubber straps, steel springs) or non-elastic levers acting with pulling, pushing, compressing, distracting or twisting power. From the Anglo-American, German, French and Italian literature of the following fifteen years I have collected the descriptions of twenty-three other active scoliosis braces, but only five of them showed marked differences from those previously described in the above-mentioned textbook. In contrast to this comparatively fertile work of orthopedic surgeons and brace makers in designing and developing new types of braces, only very few papers are devoted to a study of the theoretical mechanical bases of this part of our work. A good knowledge of these principles, however, is indispensable not only for the constructor of new braces but also for the orthopedic surgeon and brace maker in daily routine labor.

The following article is limited to the rules of construction of active body braces. It does not deal with the theory of constructing body braces for correction of deformities of the spine in sagittal plane (kyphosis), nor with braces for fixation without corrective purpose, such as given for vertebral fractures, inflammatory conditions (Pott's disease), arthritis of the spine, etc. Corresponding to the other indications, the construction of these body braces is entirely different.

From the mechanical point of view we may divide the deformity called scoliosis into four different components, which may but need not necessarily be present all together in the same case: (1) curvature of the spine itself, (2) overhang of the spine over one side of the pelvis; (3) torsion and hump formation; (4) diminution of the height of the body.

The question now arises, can we correct these different parts of the deformity by active body braces?

In order to straighten out a curve, we use the principle of three point pressure. (Fig. 1.) The most important of these three forces is power 2 which acts as the kinetic force while powers 1 and 3 are the stationary counter forces which stabilize the spine. (Fig. 2.)

Unfortunately we are unable to bring these forces to act directly upon the spine itself. We can work only through the medium of the ribs, losing herewith a considerable part of the acting force, since there is not an osseous, but only articular connection between spine and ribs. If we want to apply the pressure of the kinetic force 2 at the only effective point where it is necessary, namely at the culmination of the curvature, we must remember that the ribs have an oblique course. Therefore, the corrective force has to be located below the apex of the curve. The active pad of the scoliosis brace of an asthenic individual with a small epigastric angle must therefore be located a great deal lower in comparison with that in a patient who has emphysematic thorax of athletic type.

* From the Department of Orthopedic Surgery, State University of Iowa, Service of Arthur Steindler, M.D.

Another important rule is to direct the active pressure not in the horizontal plane, but obliquely following the course of the ribs. (Fig. 2.) The action of power 3, which means the fixation of the lower spine in the brace, is simple because of the strong connection between spine and pelvis. We need merely a pelvic construction of the brace which embraces the iliac bones tightly. The fixation of the upper spine which represents the action of power 1, cannot be obtained so firmly as at the lower end. This is due to the fact that we have no other means of fixation besides the pressure of the axillary crutches of the brace on the ribs in the axilla between anterior and posterior axillary line. The purpose of the axillary crutch is to embrace the thorax firmly, not to lift the shoulder girdle. Failure to understand this principle results in an improperly shaped axillary crutch.

Fig.1.

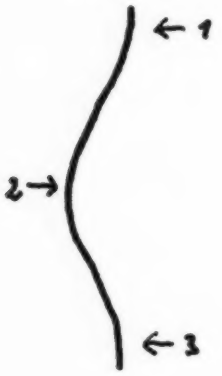
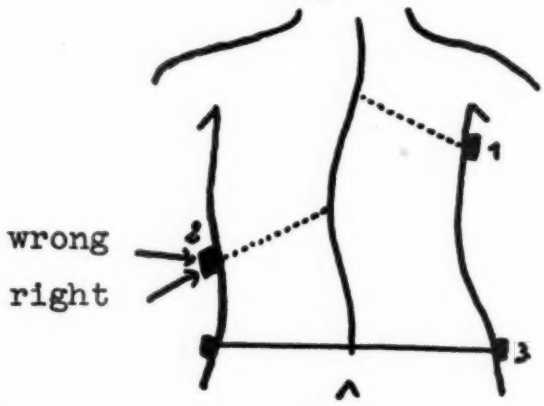
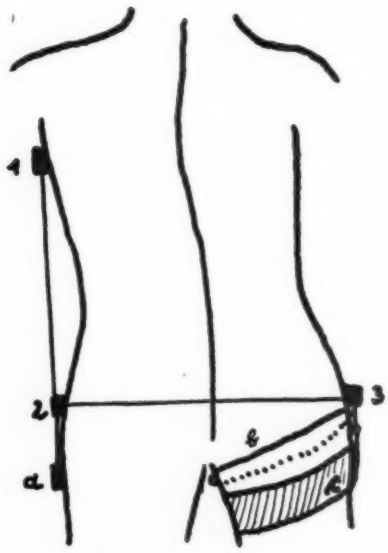


Fig.2.



If we give the crutch a shape like that shown in Figure 4, only incomplete pressure (sometimes none at all) acts upon the ribs. Instead, such a crutch brings pressure against the muscular walls of the axilla, i.e., pectoralis and teres and latissimus dorsi respectively, causing discomfort and pain to the patient. The correct axillary crutch, therefore, must be shaped as shown in Figure 5. Other important features of the pelvic construction as well as of the axillary crutches will be discussed later.

Fig.3.



a-trochanter pad
b-glutaeal strap
c-leg extension

The correction of the overhang of the body over one side of the pelvis may be achieved in two ways: first, by a pushing

power through a pad attached to an upright, ascending from the side part of the pelvic construction, or by straps attached at this upright and the paravertebral upright; and second, with a pulling power by means of a strap attached at one or two uprights ascending from the contralateral side of the pelvis construction, embracing the thorax and thus pulling it to this contralateral side. While the last construction is chosen especially for night braces, plaster or celluloid beds and plaster jackets, for day braces action by pushing power is far more convenient.

Fig. 4.

Fig. 5.

No matter which one of these two ways we choose, the important fact common to both is that the pelvic construction must fit in absolutely immovable form to the pelvis. The effect of the lever mechanism becomes entirely useless if the pelvic part tips over on the affected side. What means are used to prevent this tilting? We can use the resistance of the thigh of the affected side by a trochanter pad (Fig. 3A) attached at the lower band of the pelvic construction. This acts like the counterbalance of a scale. Furthermore, we can prevent the upward sliding of point 3 in Figure 3 by a perineal strap (Fig. 3B) on this contralateral side of the overhang. Finally and most effectively there is the method, recommended by Steindler, of incorporating one thigh, of either the overhanging or the contralateral side, in the brace. Such a leg extension, however, requires a joint at the hip.

The correction of the torsion of the spine in the sense of detorsion is the most difficult problem. To untwist the spine is impossible by exterior means. We are limited in this struggle to apply our forces on the deformity subsequent to the spinal torsion, that is, the deformity of the ribs. This can be gained by direct pressure on the hump by a pad which is attached by a lever to the pelvic part or by straps or springs, etc., attached at two uprights of the brace, usually the paravertebral and the posterior upright of the axillary crutch. The action of this appliance can be combined with the appliance for power 2 in Figure 2.

The spine, in cases of scoliosis, drops down and has to be straightened out. The most powerful means of achieving this is head extension, either by a construction similar to the old jury mast or by some kind of collar. These constructions, however, cause discomfort for the patient, especially from the cosmetic point of view, and therefore, other methods of obtaining an extension of the body have been attempted.

In former years it was believed that axillary crutches could extend a collapsed thorax. Today, as previously mentioned, we know that the mobility of the shoulder girdle is too great to allow an effect on the spine by lifting the shoulders. High

axillary crutches only lengthen the spino-scapular muscles, especially the rhomboidei, and cause an uncomfortable and ugly posture with high shoulders and short neck, sometimes even resulting in brachial neuralgia. To straighten the collapsed thorax we choose therefore another way, namely, the method of distraction. This is a traction of a snugly fitting thoracical ring or corset against the pelvic part. The success of such a procedure depends upon an absolutely firm fixation of the pelvic part of the brace at the pelvis. It is obvious that a simple belt or girdle which is often the only attachment of the brace at the pelvis never can prevent a downward sliding. Therefore an active body brace needs always two pelvic bands. One follows the iliac crest, the other crosses the tip of the greater trochanter. The upper band, following the pelvic crest, must be twisted to an oblique angle with the transverse plane of the body. Herewith a complete weight bearing support again the iliac crest can be obtained which is no less important for a body brace than the weight bearing at the tuber ischii for a long leg brace, or at the tibial condyle for a short leg brace. A proper fitting pelvic construction of an active body brace depends more than other parts of the brace upon the exact moulding of the plaster model. It follows therefore a line: sacro-iliac joint—iliac crest—swinging round the anterior iliac spine—tip of the greater trochanter—lower entrance of the sacro-iliac joint.

We know that by an active body brace a scoliosis cannot be corrected. The pressure necessary to straighten out a curved spine by pressure upon the deformed ribs has been estimated as high as 20 kg. per square cm. of skin. But what we can do is to continue the correction of a formerly rigid scoliotic spine, made mobile by the Abbott, Galeazzi, Risser or a similar method. We can also relieve the pain of intercostal neuralgia due to the compression of the intercostal nerves between the displaced ribs. A careful observance of the above given rules in construction of active scoliosis braces may help to achieve this aim.

SUMMARY

Active scoliosis braces are orthopedic appliances which, by means of elastic forces or levers, tend to decrease the scoliosis. From a mechanical point of view, the deformity consists of four different components, which can, but need not of necessity, be all present in the same case: curvature of the spine itself, overhanging of the spine over one side of the pelvis, torsion of the ribs with hump formation, and lastly diminution of the height of the body. The most commonly used devices to overcome these different malformations are described.

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CLINICO-ROENTGENOLOGIC CONSIDERATIONS OF ACUTE THORACIC EMPYEMA*

THORACIC COMPLICATIONS AND SEQUELAE IN THE NON-TUBERCULOUS FORM

SAMUEL GEORGE SCHENCK, M.D. AND LEW A. HOCHBERG, M.D.

Associate Attending Radiologist, Jewish Hospital — Assistant Attending Surgeon, Jewish Hospital

BROOKLYN, NEW YORK

DIFFICULTY and uncertainty are often experienced in eliciting physical signs in the chest of a patient with post-operative empyema. Therefore, much reliance has come to be placed on the Roentgen examination to follow the progress of the empyema and to discover complications as they may arise.

Roentgenograms taken at frequent intervals portray not only the comparative difference in the thorax following the evacuation of pus, but also the condition of the structures upon which the clinical course depends. Thus, the presence of retained fluid, free or encapsulated in the pleural space or interlobar fissures, the position and location of the drainage tube, the degree of expansion of the lung and its appearance, the changes in the pleura, the status of the diaphragm, the position and contour of the heart and mediastinal structures, and the appearance of the resected rib or ribs are but a few of the important considerations which are visualized by the Roentgen ray. There is no better method at our disposal to determine the status of the empyemic patient than serial roentgenography.

Most of the complications and sequelae in acute empyema are readily demonstrable on the roentgenograms of the chest. The affected structures and their changes which are seen roentgenologically may be tabulated as follows:

A. Pleura:

1. Retained fluid, free, encapsulated or interlobar.
2. Thickened pleura and pleural adhesions.
3. Secreting or pyogenic membrane.
4. Subacute and chronic empyema.
5. Bilateral empyema.
6. Pyopneumothorax, free and encapsulated.
7. Persisting pneumothorax; free, encapsulated and pocketed.
8. Fibrosis of the pleural space.
9. Fibrinous bodies in the pleural cavity.
10. Calcification.

B. Lung:

1. Collapsed or atelectatic lung.
2. Solidification and cornification of the lung.
3. Incomplete and irregular lung expansion.
4. Pleuropulmonary adhesions.
5. Pneumonia, active, unresolved, or recurrent.
6. Lung abscess and gangrene.
7. Bronchopleural fistula.
8. Chronic interstitial pneumonitis.

C. Mediastinum:

1. Displacement of the mediastinal contents.
2. Mediastinal adhesions.
3. Suppurative mediastinitis.
4. Pleuro-esophageal fistula.

D. Heart:

1. Cardiac displacement (dextrocardia).
2. Pleuro-pericardial adhesions.
3. Suppurative pericarditis.
4. Cardiac hypertrophy.

E. Diaphragm:

1. Adhesions.
2. Elevation of the diaphragm.
3. Diaphragmatic fluid and encapsulations.

* From the Departments of Radiology and Surgery, Jewish Hospital.

4. Subphrenic collection.
5. Abdominal abscesses and peritonitis.
6. Lumbar and psoas abscesses.

Fig. 1. Retained fluid in the lower half of the left chest. The dense opacity obliterates the costophrenic sinus and obscures the diaphragmatic leaf. The upper level of the fluid is highest in the axilla and curves downward as it passes mesially.

F. Rib:

1. Resected rib or ribs.
2. Osteogenetic repair of the resected rib.
3. Osteomyelitis of rib or ribs.
4. Persisting sinus formation or fistula.
5. Narrowing of the intercostal spaces.

G. Spine:

1. Curvature of the dorsal spine.
2. Osteomyelitis.

H. Soft Tissues of the Thorax:

1. Empyema necessitatis.
2. Fistulous tracts.

I. Foreign Bodies:

1. Drainage tube.
2. Foreign bodies in the pleural sac.

PLEURAL COMPLICATIONS

The Roentgen examination is the most satisfactory method to determine the presence or absence of retained fluid. (Fig. 1.) It becomes difficult only when the fluid is encapsulated or pocketed, or when the pleura becomes sufficiently thickened to approach the density of fluid. Although the differentiation between thickened pleura and retained fluid is often not difficult, nevertheless there are occasions when the two cannot be easily separated. The differentiation is particularly trying when the retained fluid is obscured by an overlying thickened pleura. (Fig. 2.) Ordinarily, a thickened pleura casts a diffuse haze, whereas fluid produces a rather dense opacity. (Figs. 3 and 4.) Roentgenoscopy in conjunction with erect and prone films usually determines the presence or absence of fluid, especially when a small amount of air is also present in the pleural space. The presence of fluid may be ascertained by its mobility with a change in the patient's posture, whereas in pleural thickening this finding does not take place. The presence of retained fluid is often demonstrable as a strip of opacity extending along the axillary gutter. This shadow is frequently overlooked because it is obscured by the ribs in the axillary region and may be clearly demonstrated by roentgenograms in the lateral prone position. (Fig. 5.) It must be remembered that less than 250 c.c. of fluid may be insufficient to cast a Roentgen shadow or may lie hidden in the sulci of the diaphragm.

The localization of pockets of pus can be more satisfactorily accomplished by Roentgen means than by any other method at our disposal. (Figs. 6 and 7.) Their number, location and size (Fig. 8) are readily visualized and the information obtained is of inestimable value in determining the most favorable operative approach and procedure. In empyema involving the left pleural space, one must not overlook the possibility of a collection behind the heart which may obscure the pathology. (Fig. 9.) Pus encapsulated in the interlobar fissures assumes the position and direction of the interlobar spaces. (Fig. 10.)

Adhesions which are clearly demonstrable on the roentgenograms may interfere with proper drainage, diaphragmatic mobility, reexpansion of the lung and often accounts for residual intercostal pain. (Figs. 11 and 12.) Air in the pleural cavity

may come through the thoracotomy wound or the drainage tube, from needle aspirations of the chest, from the bronchopleural fistula or may be the result of a gas-producing organism. Later, the presence of a fistulous tract through the site of operation may account for a persistent pneumothorax. Not unlike fluid, air may be free in the pleural space or encapsulated by adhesions, frequently resulting in a localized pneumothorax or hydropneumothorax at the site of exit of the drainage tube. (Figs. 13, 14 and 15.)

In addition to the presence of foreign bodies discussed below, the pleural cavity may show such extraneous shadows as fibrin masses. As a sequel to empyemas of long duration in which the pleural layers become thickened and enclose inspissated pus, the pleura may show calcific deposits. Such changes are recognized only on the films.

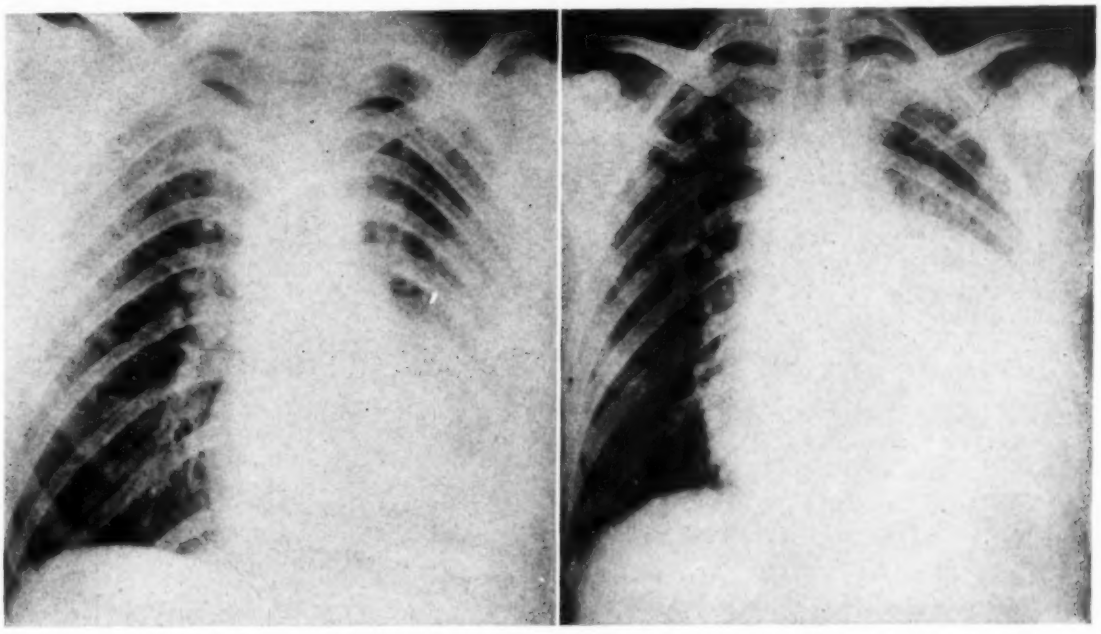


Fig. 2. Retained pleural fluid with overlying thickened pleura. Three months previous to this study, the first thoracotomy was performed for acute empyema. An empyema necessitatis developed, requiring a second thoracotomy with rib resection. This roentgenogram shows not only the oblique upper limit of the fluid traversing the chest, but also a narrowing of the intercostal spaces as a result of pleural fibrosis resulting from the chronicity of the pathologic process. Note the compensatory emphysematous changes in the right lung as evidenced by the widened interspaces and the low diaphragmatic dome.

Fig. 3. Residual thickened pleura before fibrosis and contraction of the hemithorax have taken place. Although the density of the shadow is almost as pronounced as that of the fluid, yet the absence of the oblique upper border of the shadow, as described in Figure 1, and the gradual fraying of the opacity superiorly tend to exclude the diagnosis of fluid. Note the absence of cardiac and tracheal displacement to the opposite side, as one might expect in a case of fluid occupying two-thirds of the chest.

Because the pleural sac is frequently obliterated following infection, recurrence of empyema at the same site rarely occurs. Due to incomplete evacuation or absorption, there may be a recurrence of symptoms and an increase in the amount of fluid. In this way a relapse or an acute exacerbation of a chronic empyemic process (Figs. 2 and 16) ensues. The recurrence is due to inadequate drainage, undiscovered localized collections, free or encapsulated, or the development of empyema involving a portion of the pleura which has not been previously affected. Not infrequently, prolonged drainage from an empyemic cavity or reaccumulation of fluid in the chest is due to a secreting or pyogenic membrane on the pleura. Such membranes may be an

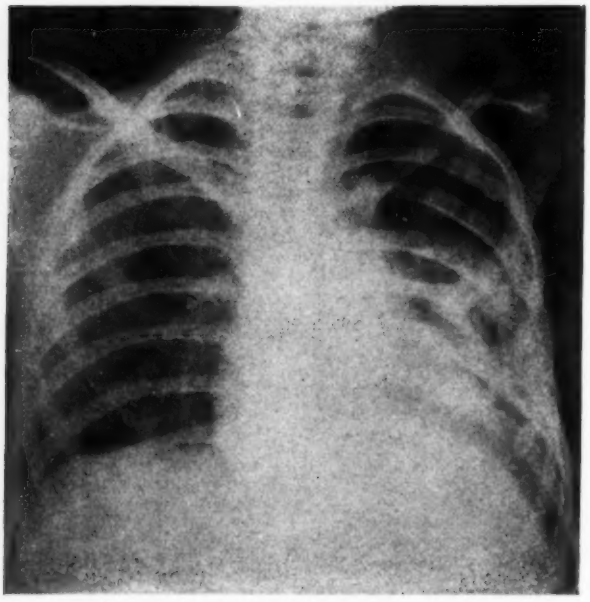


FIG. 4. The diffuse haze in the left lower chest represents thickened pleura. No underlying fluid is observed, as the lung, costophrenic sinus and diaphragmatic leaf are visible through the haziness. All of the factors secondary to thickened pleura of long standing are present: the intercostal spaces are narrowed as compared to the opposite side; the heart is slightly drawn to the left; and the spine presents a slight curvature to the contralateral side. These findings are due to pleural fibrotic contraction. Also note how the resected rib has repaired with a central cloaca.

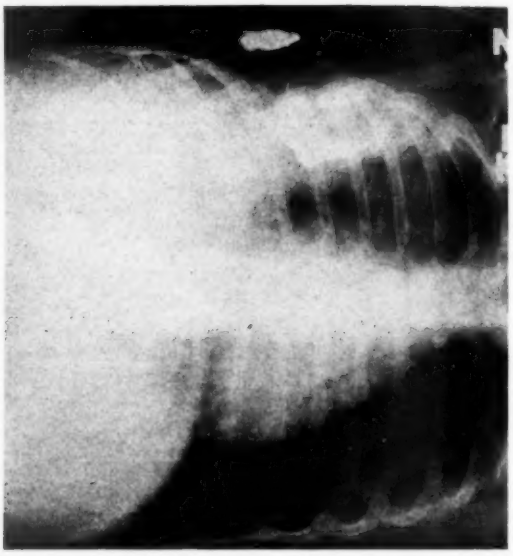


FIG. 5. Roentgenogram in the lateral prone view with the right thorax up, showing a strip of opacity along the right axillary gutter, indicative of retained fluid. Iodized oil has been injected into this space through the thoracotomy wound. Observe the healing of the old resected ninth rib in the axillary area.

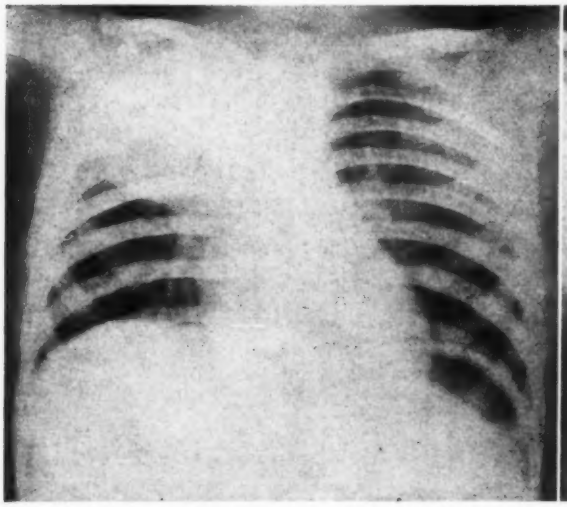


FIG. 6. Encapsulated fluid in the right upper chest. The localized collection of fluid presents a sharp curvilinear lower border. No cardiac or mediastinal displacement to the opposite side obtains.

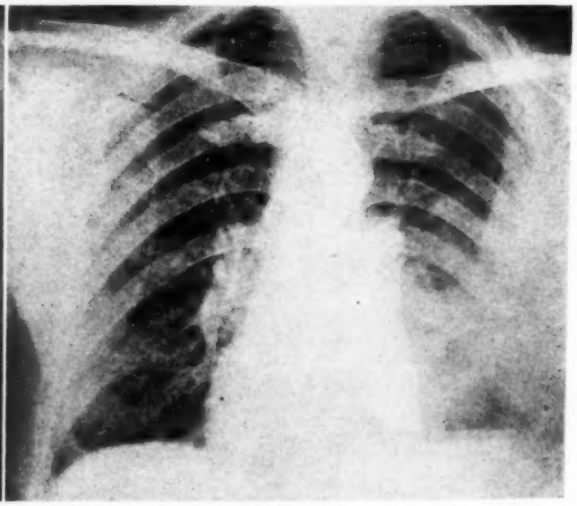


FIG. 7. Encapsulated fluid along the left axilla. Observe the sharply defined mesial border of the collection.

inch in thickness. Although the diagnosis cannot be absolutely made by Roentgen study, it may be inferred with reasonable certainty by the visualization of a thickened pleura, the incomplete expansion of the lung, and by the absence of other causes. (Fig. 12.) Clinically, it is recognized that there is a greater tendency for pleural thickening in the pneumococcic empyemas than in the streptococcic forms.

In some instances, pleural suppuration may extend to the pericardium, peritoneum or mediastinum, and to the opposite pleura. (Fig. 17.) Such extensions, although uncommon, occur most frequently in streptococcic empyemas.

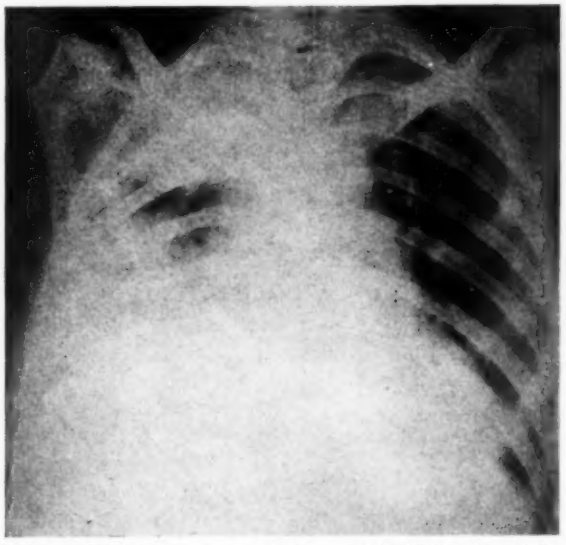


Fig. 8. Multiple encapsulations of fluid in the right chest. Roentgenogram reveals the presence of retained fluid in the lower half of the thorax and a large collection in the upper region.

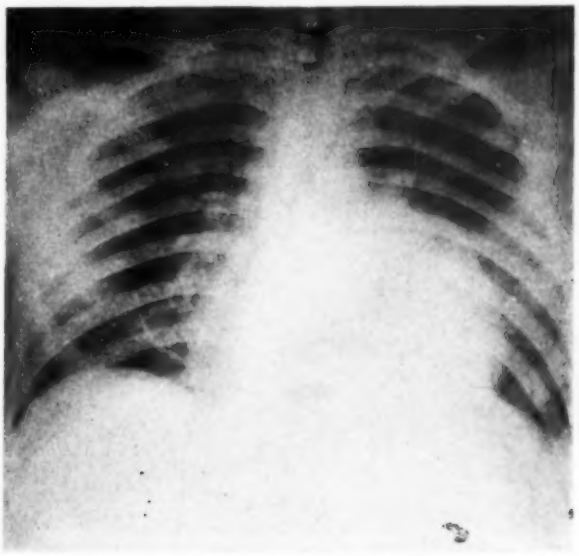
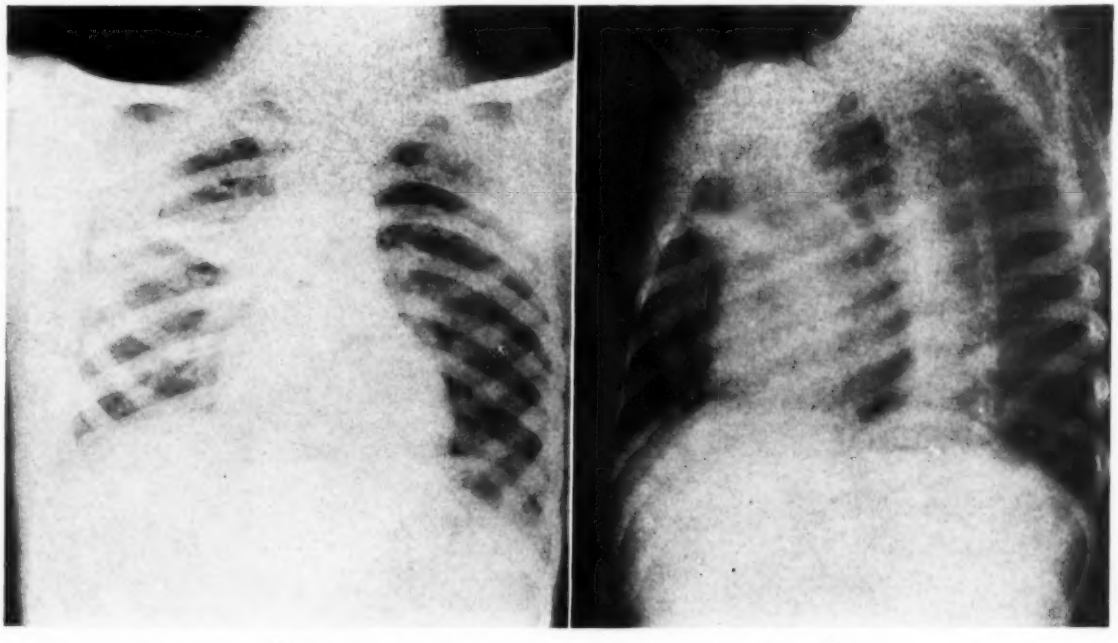


Fig. 9. A rim of encapsulated empyema is visible beyond the left cardiac shadow. The remainder is entirely obscured by the heart.



A B

Fig. 10. A, the triangular area of opacity traversing the right pulmonic field is a collection between the upper and middle lobes. B, same in the right oblique view.

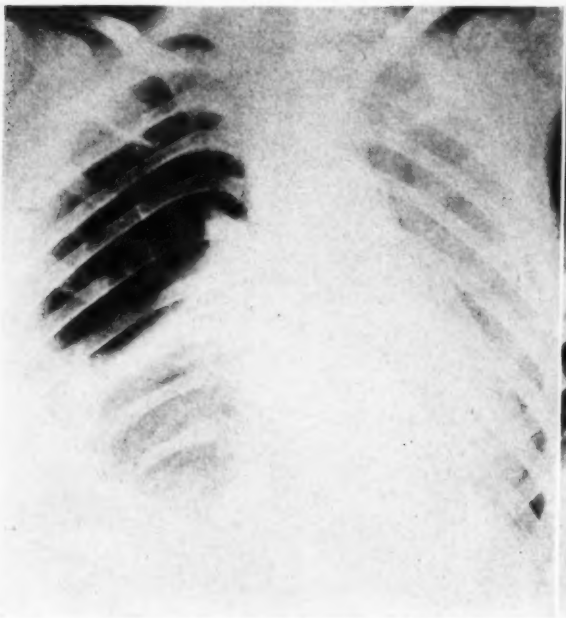


FIG. 11. The right chest shows multiple pleural adhesions which interfere with the expansion of the lung.

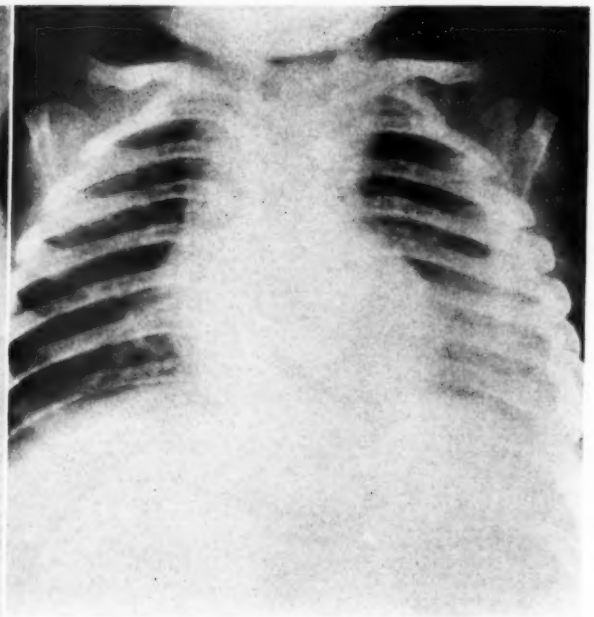


FIG. 12. A residual pneumothorax persists along the left axilla, associated with a thickened visceral pleura, preventing complete lung expansion. The diffuse haze in the left lower chest also represents a thickened pleura with a pyogenic membrane. This infected surface accounts for the frequent attacks of fever over a period of many months.

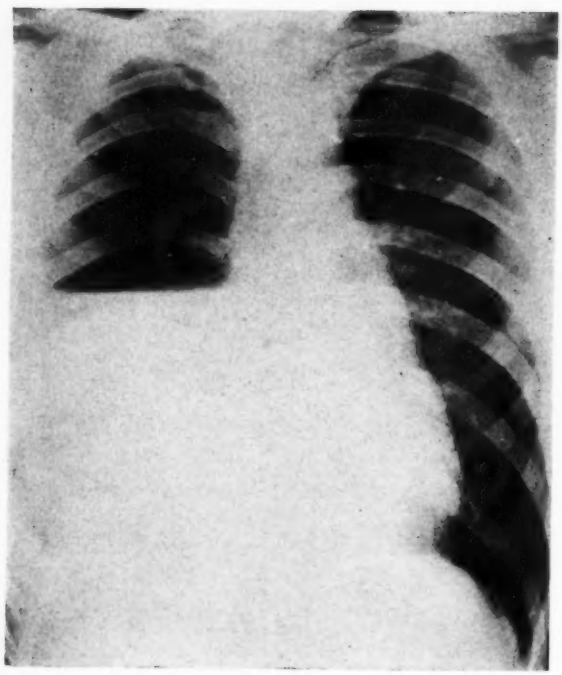


FIG. 13. Pyopneumothorax occupying the entire right hemithorax. With the patient in the erect posture, the horizontal fluid level with air above is clearly shown.

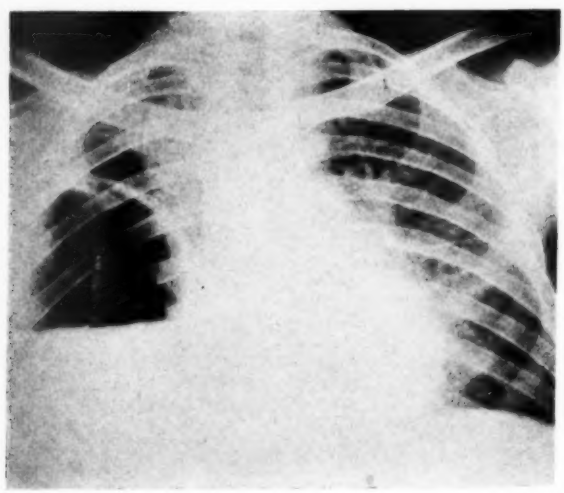


FIG. 14. Encapsulated pyopneumothorax presenting a localized collection of pus and air. A horizontal fluid level is shown.

PULMONARY CONSIDERATIONS

The Roentgen appearance of the lung offers considerable information. With hemi-

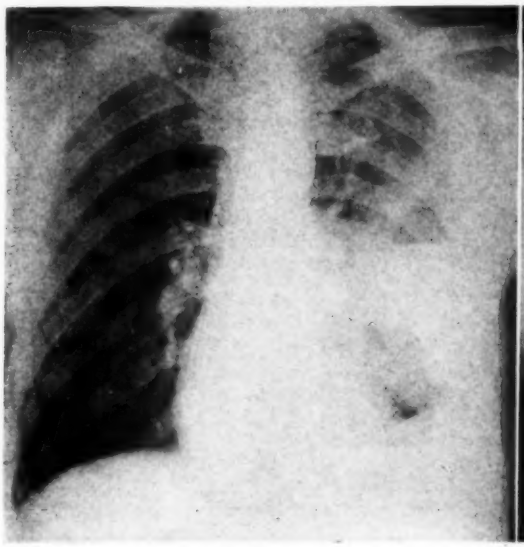


Fig. 15. Encapsulated pyopneumothorax in the left axilla. Observe the adhesions to the pleura about the pus pocket.

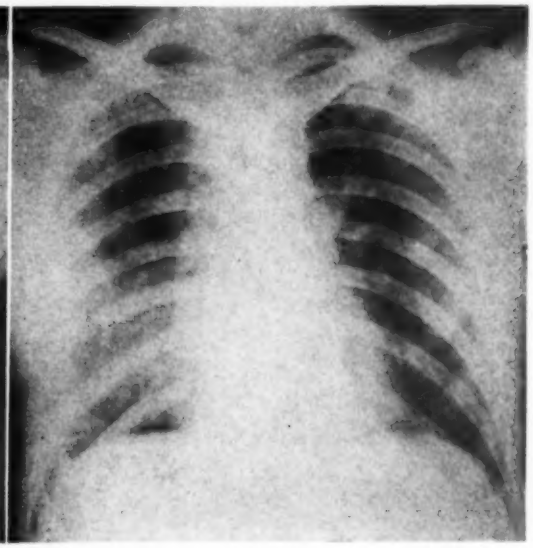
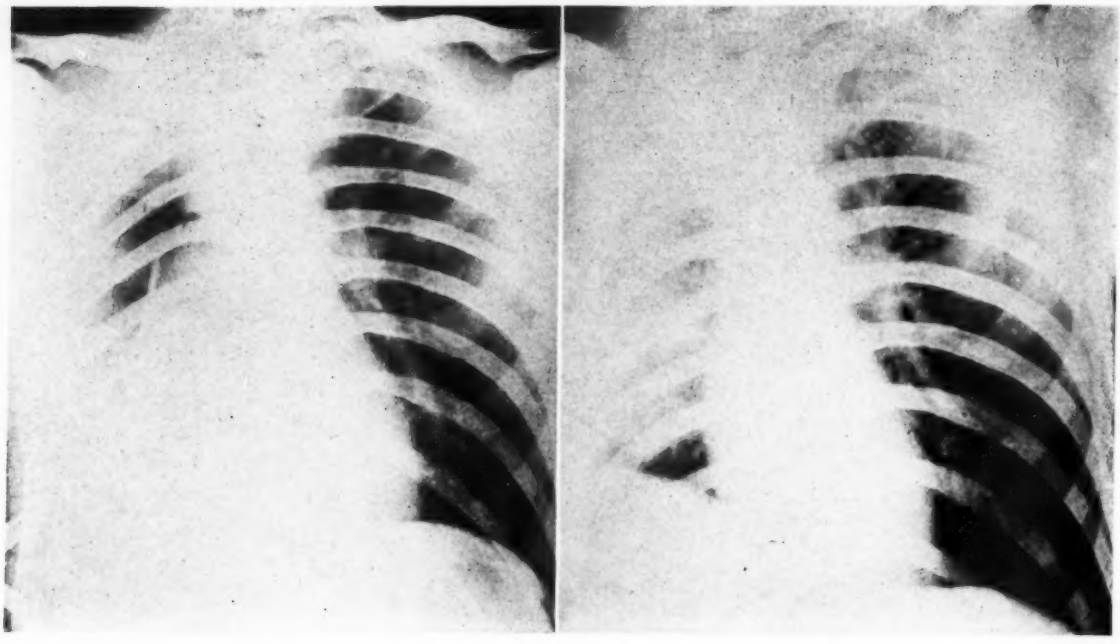


Fig. 16. Chronic empyema of the right chest. The pathologic process failed to clear up and necessitated a thoracoplasty eight months later.

thorax full of fluid, the lung is compressed toward the hilus, provided no firm adhesions bind it to the thoracic cage. As the pus is evacuated after surgical intervention, the lung reexpands. Not infrequently, pulmonary expansion is complete, except for a small area at the costophrenic sinus and for a narrow strip along the extreme axillary border. Although all the fluid has been evacuated, these spaces may fill in with fibrous tissue which persists. If air occupies these areas, it is eventually absorbed, provided no additional air is drawn in through the thoracotomy wound. As a result, the lung completes its expansion. If the lung expands unevenly, it may enclose pus and air, forming pockets of pyopneumothorax.

There are occasions when the lung fails to expand. Ordinarily, with a chest full of fluid, it lies fully collapsed at its root, and if allowed to remain so for a long time, may be partially or wholly incapable of reexpansion. Thus, solidification or cornification of the lung in part or in its entirety may take place due to chronic interstitial changes. To obliterate the pleural cavity, all the structures of the chest may be affected; the heart and mediastinum are drawn to the affected side, the intercostal spaces become narrowed, the diaphragm is drawn upward and fixed, the spine undergoes a lateral curvature and any remain-

Fig. 17. Roentgenogram reveals bilateral empyema with pericardial fluid. The process extended from the right pleural cavity to the pericardial sac and then to the left pleura.



A B

C

FIG. 18. A, the right lung appears partially collapsed due to thickened visceral pleura. Note also the thickened parietal pleura and fibrosis. This fibrosis is further evidenced by a contraction of the right chest. The partially expanded lung also shows early chronic fibrotic changes. There are compensatory emphysematous changes in the contralateral side. B, six months later, the lung appears somewhat more expanded and fills the shrunken hemithorax. The pronounced contraction of the right hemithorax is now more clearly portrayed. The haziness produced by the thickened pleura, the chronic pulmonary interstitial changes, the elevated diaphragmatic cusp and the dislocation of the heart and trachea to the affected side are readily visualized. Note the marked compensatory emphysema of the left chest. C, the final result, following total thoracoplasty with complete recovery.

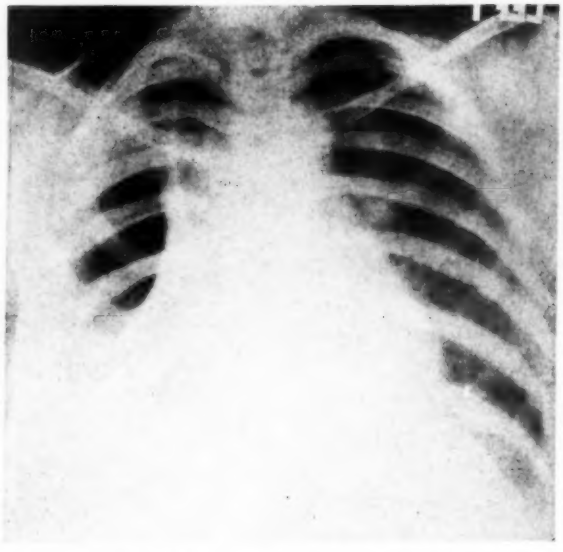


FIG. 19. Chronic pneumothorax persisting for five months due to four bronchopleural fistulae found on operation.

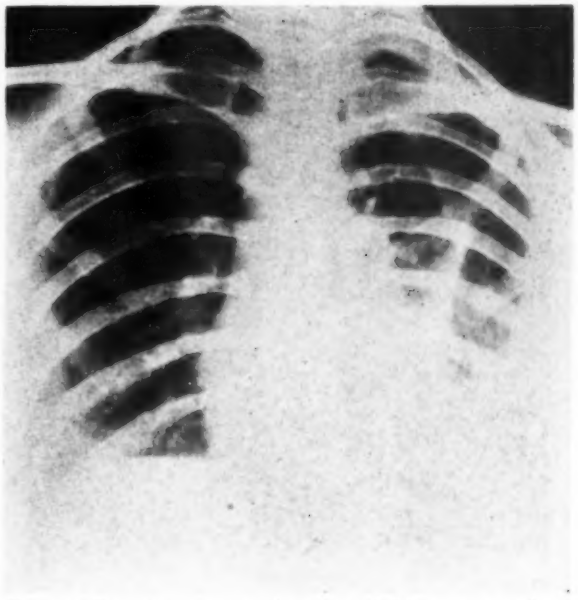
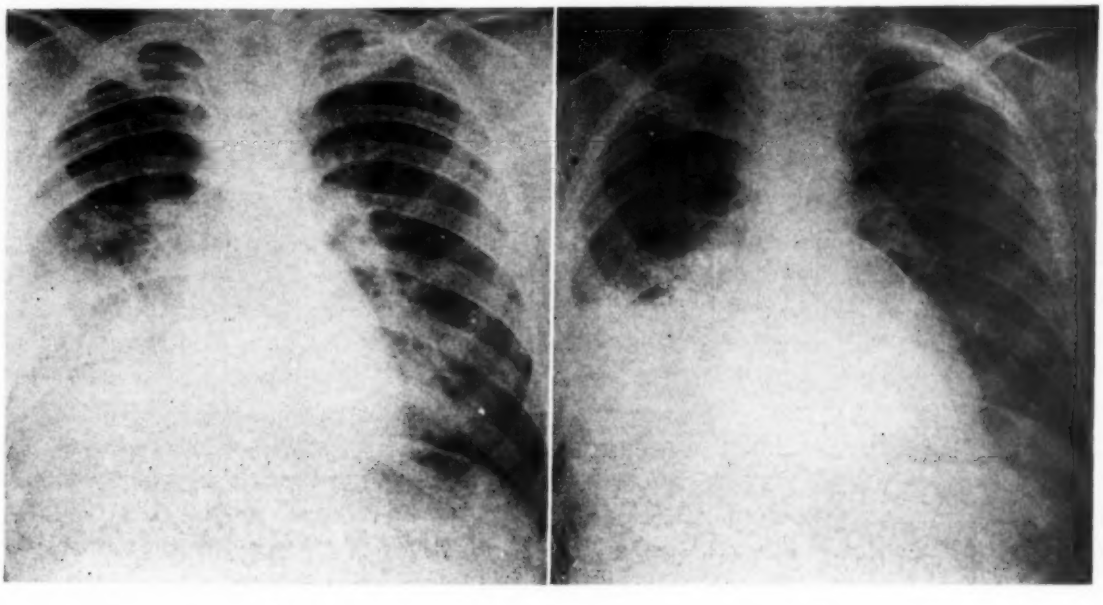


FIG. 20. Roentgenogram shows partial lung expansion. The lower part of the pulmonary field appears infiltrated. There is evidence of retained fluid and thickened visceral pleura. For two months the lung failed to expand until three days after this roentgenogram the patient coughed up a thick mucous plug one-half inch in diameter. Subsequently the lung showed less infiltration and progressive expansion.



A B

FIG. 21. A, pulmonary abscess is revealed in the expanded right lung after drainage of the empyemic process. B, the same case showing the abscess cavity filled with iodized oil.

ing space is infiltrated with connective tissue. (Fig. 18.) With these changes a compensatory emphysema of the contralateral thorax takes place.

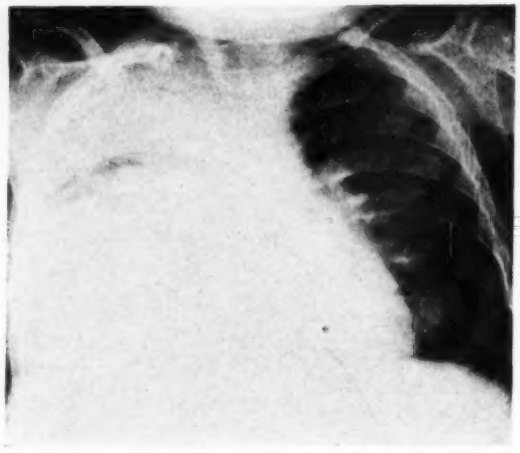


Fig. 22. Post-operative empyema with no improvement following three thoracotomy operations. The right chest shows multiple pleural and pulmonary pus pockets accompanied by considerable chronic fibrotic changes in the lung and pleura. Note the resulting contraction of the hemithorax. The interspaces are narrowed and the heart and trachea are drawn to the affected side.

Again, the failure for the lung to expand, after the pleural fluid has been released, may be due to air replacing the fluid in the chest. Such a condition obtains in bronchopleural fistula. The resulting persistent pneumothorax keeps the lung collapsed or partially expanded, depending on the pressure in the pleural cavity, the size of the fistulous opening, and the presence of pleuropulmonary adhesions. (Fig. 19.)

Another cause for incomplete lung expansion is atelectasis due to bronchial obstruction produced by a mucous plug. (Fig. 20.)

The causes of a fistulous communication between the pleura and lung are: (1) rupture of a peripheral lung abscess; (2) rupture of an expanding friable lung; (3) pressure necrosis of the lung produced by the drainage tube; and (4) a spontaneous pneumothorax.

The expanding pulmonary structure often reveals itself with sufficient clarity to portray the presence of infiltration or other pathology. An active or a recurrent pneumonic lesion may be demonstrated, or the patient's persistent cough with a low-grade type of temperature may be

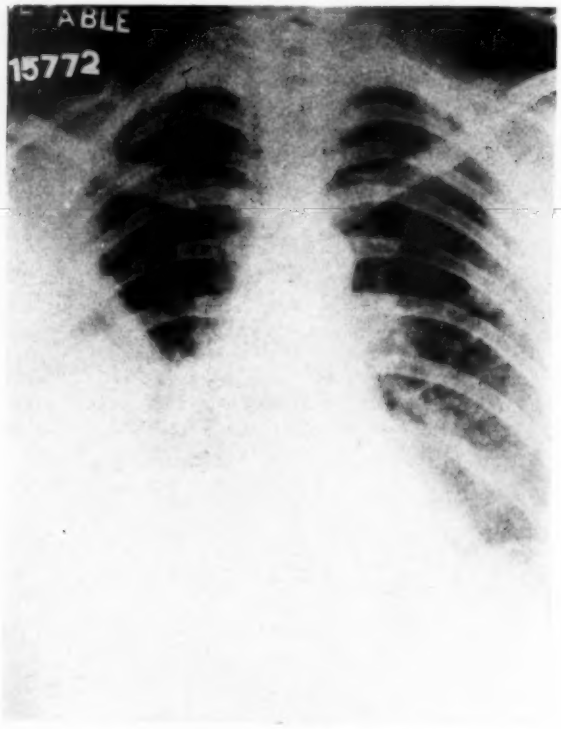


Fig. 23. Encapsulated pocket of pus which ruptured into a bronchus. Three weeks following the onset of influenzal bronchopneumonia, the patient developed an encapsulation which was not drained nor aspirated. Suddenly, she began to expectorate purulent material. The above bedside roentgenogram taken at this time shows not only the encapsulation along the lower axilla and the air pocket, but also the infiltrative process in the adjacent lung where the perforation took place. Findings were confirmed at operation.

accounted for by the presence of unresolved pneumonia or delayed resolution. Pulmonary suppuration or abscess formation or gangrene may be observed in the lung bed, the pathology being primary or secondary to the empyemic lesion. (Figs. 21 and 22.) Even on post-mortem examination, it is sometimes difficult to ascertain whether the empyemic process perforated into the lung and secondarily caused single or multiple pulmonary abscesses or whether the lung suppuration ruptured into the pleural sac and produced an empyema. A lung perforation due to empyema may

cause a sudden paroxysm of cough with the expectoration of pus, the quantity varying with the size of the communication. Single or multiple fistulous tracts between the pleura and the bronchi may develop and a persisting pneumothorax may ensue therefrom. If the patient recovers and the suppuration in the lung is evacuated, chronic interstitial pulmonary changes are likely to follow. (Fig. 23.)

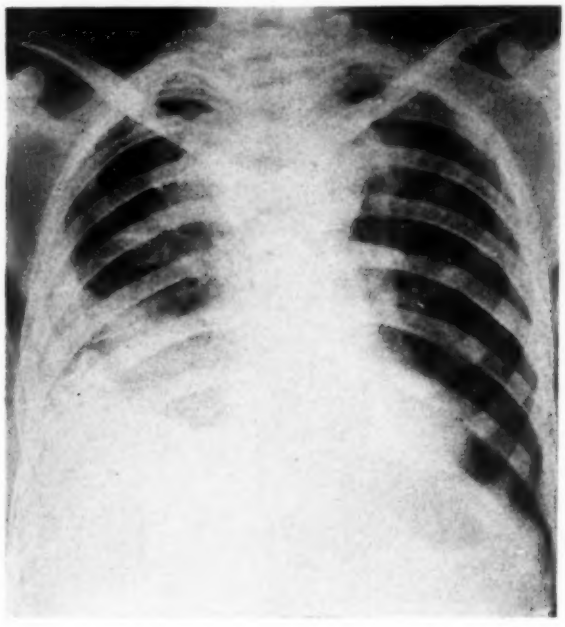


Fig. 24. Roentgenogram shows a large superior mediastinal abscess, which followed a right thoracic empyema.

MEDIASTINAL FACTORS

The shift of the mediastinal structures with unilateral pleural effusion of sufficient degree is well known, unless the mediastinum is fixed. As the fluid and air drain off, the structures tend to return to their normal position. Later, due to adhesions or contracting pleural fibrosis, the mediastina may be drawn to the affected side and produce cardiac or respiratory embarrassment.

Additional complications are the presence of mediastinal adhesions and fluid. The latter is usually encapsulated (Fig. 24) and presents one of the most serious considerations although infrequently recognized during life. The localization of a pocket of pus in the mediastinum is best accomplished by Roentgen means, which guides the surgeon in his operative procedure. It may be difficult to decide whether the empyema developed first and subsequently invaded the mediastinum, or whether the empyema resulted from rupture of a mediastinal abscess into the pleura, the latter occurrence probably being somewhat more frequent than the former. A preexisting acute cervical and retropharyngeal adenitis usually suggests that the mediastinum was involved before the pleura, and that the mediastinal abscess ruptured into the pleural cavity. Mediastinal abscess following empyema is generally streptococcal in origin.

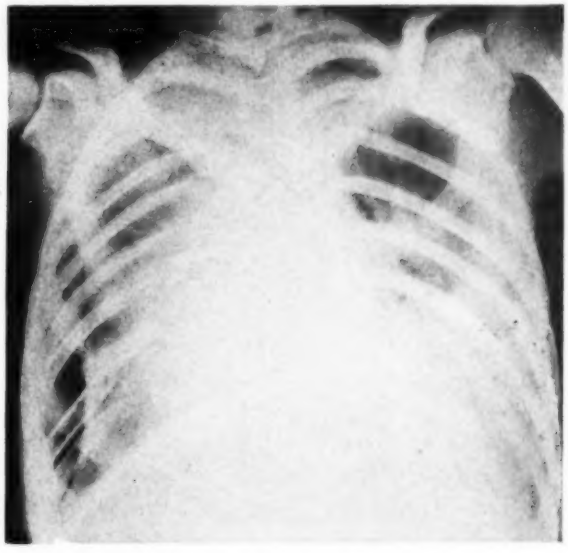


Fig. 25. Same case as Figure 17. There is evidence of marked cardiac enlargement in all diameters. The findings represent pericardial suppuration with septic involvement of the myocardium and endocardium secondary to right thoracic empyema. Note the spread of the suppurative process to the left pleura.

Roentgenologically, mediastinal adhesions are manifested by a fixed mediastinum with broadened borders, which obscure or obliterate the normal delineations. Clinically, the condition presents a fixed mediastinum and trachea.

The effect of mediastinal pathology on the adjacent important structures is often demonstrable on the screen and films. Tracheal and esophageal displacement and deviation due to compression by medias-

tinal fluid may account for extreme dyspnea and dysphagia, respectively. The mediastinal abscess may perforate into the esophagus with the formation of a fistulous tract.

Fig. 26. A broad dense band is seen passing from the diaphragm to the pleura about a pocket of pus. Roentgenogram shows a lateral view of an encapsulated pyopneumothorax. Same case as shown in Figure 15.

CARDIAC COMPLICATIONS AND SEQUELAE

Like the mediastinum, the position of the heart may be notably affected by empyema. With massive effusion, the heart is shifted to the opposite side, only to return to normal after the fluid is evacuated. Occasionally, however, as with the mediastinal structures, the heart may later be drawn to the affected side by dense fibrous adhesions and pleural thickening. (Figs. 18B and 22.) With the original pathology in the right hemithorax, a secondary dextrocardia ensues. The cardiac contour itself may assume an irregular outline with tent-like elevations due to pleuro-pericardial bands. These factors may seriously interfere with cardiac function.

Secondary invasion of the pericardial sac (Fig. 17) probably occurs more frequently than is realized. Subsequent hypertrophy of the heart with enlargement of all its chambers has been recorded. Such findings are recognized roentgenologically on comparative periodic teleoroentgenographic examinations. (Fig. 25.) Purulent pericarditis following empyema is usually of streptococcic origin.

Fig. 27. Osteomyelitis in the resected eighth and ninth ribs following drainage of left thoracic empyema. Note the irregularity and osteolytic changes in the affected ribs which show some evidence of involucral deposits. The diaphragmatic cusp is elevated and irregular due to adhesions.

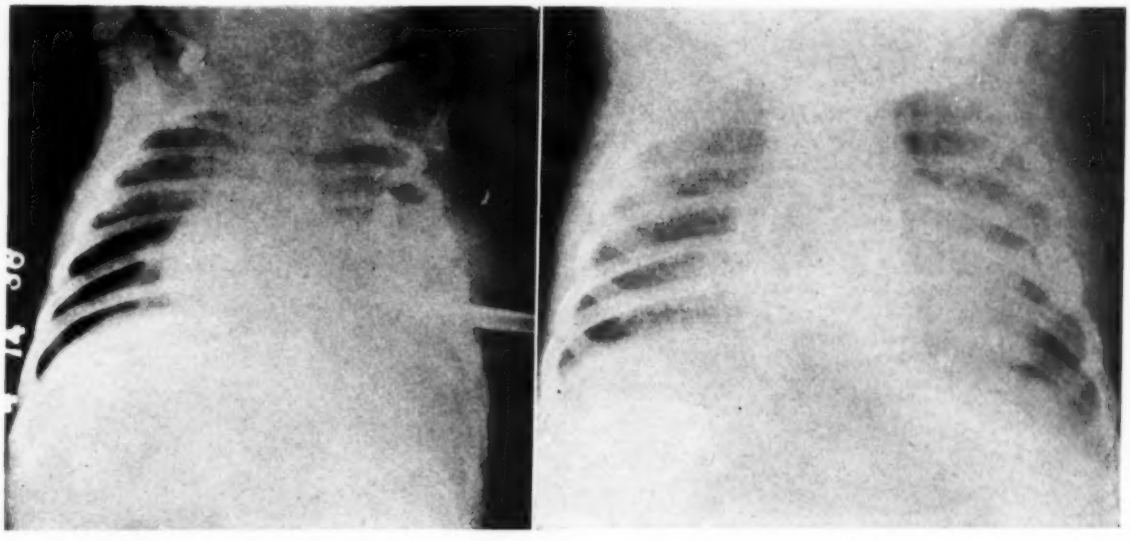
DIAPHRAGMATIC COMPLICATIONS

The diaphragmatic cusp may be fixed, elevated and tented instead of smooth and dome-shaped. Diaphragmatic pleurisy invariably accompanies the presence of pus in the pleural space. The affection

involving the cusp may entirely resolve as in the remaining pleural cavity or residual pathology may persist indefinitely.

COSTAL CONSIDERATIONS

By study of the resected rib in the films, one notes whether there is ample opening



A B

Fig. 28. A, left empyema with intercostal drainage (no rib resection). The tube which lies above the level of the fluid is also bent or kinked, resulting in poor drainage. B, roentgenogram shows chronic osteomyelitic changes of the left fifth rib due to pressure necrosis. There was no rib resection.

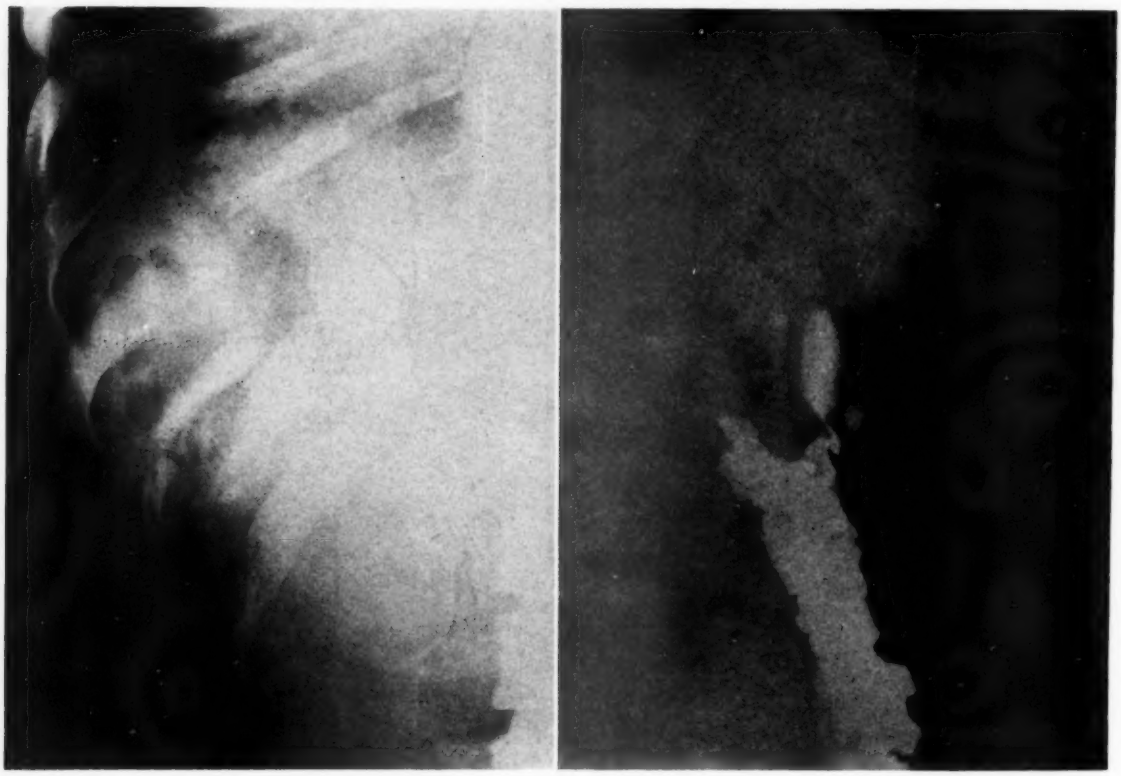
Diaphragmatic adhesions, commonly observed in the costophrenic or cardiophrenic sulci, produce a limitation of the free excursion of the retracted cusp. The loss of its smooth dome-like surface due to angulations or tenting is also the result of adhesions or bands. (Figs. 15 and 26.) It should be borne in mind that small collections may lie in the sulci and be obscured by the diaphragmatic dome. Occasionally, the empyemic lesion spreads by direct extension through the diaphragm and collects in the subphrenic space. As a result, it causes an elevation and immobility of the cusp with a slight flattening of the dome. However, it is decidedly more common for an abdominal infection such as peritonitis, liver abscess or subphrenic collection to permeate the diaphragm through the lymphatics or perforate the cusp and secondarily invade the pleura. An abdominal abscess, originating in the pleura, may perforate the stomach, the intestines or the kidney, and may even extend along the spine to the iliac fossa and simulate psoas or lumbar abscess.

for drainage, the appearance of the free margins of the rib and the presence of loose fragments of bone. Preoperatively, the roentgenogram indicates which rib should be removed to insure adequate dependent drainage. Post-operatively, it reveals whether the proper rib was removed. A not infrequent costal complication is osteomyelitis of the resected rib. (Fig. 27.) This is determined radiographically by the irregularity and worm-eaten appearance of its free margins with or without the presence of a sequestrum.

Osteomyelitis may also develop in an unresected rib. In intercostal drainage, the pressure of the tube on the rib may produce a periostitis with ensuing osteomyelitic changes. (Fig. 28.)

The progress of repair of an osteotomy is readily portrayed on the roentgenograms. The atrium is bridged by an irregular osteogenic overgrowth which in time closes the gap more or less completely. The end result of this closure will depend to a great degree on how carefully the surgeon stripped the periosteum at the

time of operation. Not infrequently, a small gap or vacuole remains in the regenerated rib, a finding which must not be confused with bone destruction. (Figs. 4 and 5.) This vacuolar area presents smooth and clear-cut margins and so may be distinguished from the fuzzy, moth-eaten appearance in osteomyelitic disease.



A B

FIG. 29. A, chronic osteomyelitis affecting the lower four ribs following empyema. B, roentgenogram shows the large fistulous tract filled with iodized oil extending from the lumbar region, where the sinus perforated, to the infected ribs. Observe the multiple fistulous ramifications passing to the many active foci in the involved ribs.

The extent and size of a persistently draining sinus tract may be visualized by the injection of iodized oil. (Fig. 29.) If the opaque oil leads to the resected rib or ribs, the presumption is that an infective process involves the costal structures; however, should the oil pass directly into the pleural space, other causes for persistent drainage must be investigated.

A final consideration in costal changes due to empyemic infection is the narrowing of the interspaces with a diminution in volume of the affected hemithorax as a late sequel to chronic fibrotic changes in the pleura. In advanced cases, the ribs may become shingled with complete obliteration of the intercostal spaces. By the same token, a widening of the interspaces in the contralateral side is the result of a compensatory emphysematous alteration. (Figs. 2, 3, 18 and 22.)

SPINAL CHANGES

Due to the pleural fibrosis and contraction resulting from pleural suppuration and terminating in obliteration of the pleural cavity or the formation of dense bands of adhesions, the thoracic spine may show a varying degree of kyphoscoliosis. (Fig. 18B.) The scoliotic element of the deviation usually predominates. The shift of the heart and mediastinal structures to the affected side with the elevation and fixation of the diaphrag-

matic cusp usually accompanies the scoliosis.

The marked shrinkage of the entire hemithorax which is accompanied by spinal deviation and drooping of the shoulder is more evident in young subjects with less resistant thoracic walls. This shrinkage also follows where there has been partial solidification of the lung.

FINDINGS IN THE SOFT TISSUES OF THE CHEST

A soft tissue swelling in the wall of the thorax may indicate the presence of an empyema necessitatis. The usual sites are in the parasternal region and in the fifth interspace outside the nipple line. The perforation may be single or multiple and is more apt to develop in the streptococcic rather than the pneumococcic empyemas. Sinus tracts or fistulae are sometimes observed in any part of the chest wall, and occasionally may extend along the muscle planes to the loin, simulating a psoas or lumbar abscess. (Fig. 29B.) They may be visualized on roentgenograms taken after the injection of iodized oil indicating their size, direction and arborization. Another complication occasionally observed in the soft tissues is the presence of subcutaneous emphysema. This may be due to the trauma of surgical intervention or secondary to anaerobic infection.

Perforation of the empyema may produce caries of the ribs and necrosis of the soft parts; even fatal hemorrhage may result from erosion of an intercostal vessel. Except for the costal destruction, these changes are not as a rule demonstrable on the *x*-ray films. By the extension of the pleural infection to the intrathoracic veins or the endocardium, metastatic abscesses to the brain, lung and other organs sometimes occur.

EXTRANEOUS CONSIDERATIONS

The main extraneous shadow in the study of post-operative thoracic empyema is the drainage tube, which must be patent and adequate in size and properly placed. Its relation to the fluid in the chest is all important to insure proper drainage. This can be best determined by roentgenographic study. (Fig. 28A.) The tube should lie at the dependent portion of the fluid, so that gravity may aid drainage; any kinking of the tube will interfere with proper evacuation of pus. Occasionally, it happens that the tube is lost in the chest. It can be readily portrayed lying in the pleural sac and recovered by the introduction of a uterine dressing forceps under fluoroscopic control or by endoscopic means.

In addition to the drainage tube, other extraneous substances may be introduced into the chest and accidently left behind. A gauze sponge in the pleural cavity, resulting in prolonged drainage with a persistent sinus cannot be revealed on the roentgenograms. Iodoform gauze, however, casts a definite shadow which is readily recognized. A strip of adhesive plaster accidently left on the patient's skin may cast a roentgen shadow and has been mistaken for a foreign body in the pleural cavity (resembling a strip of gauze).

CONCLUSIONS

The importance of serial roentgenograms to determine the exact status of the empyemic patient becomes at once evident when it is realized that roentgenography is the only accurate method at our disposal for determining the progress of the disease and the presence of intrathoracic complications and sequelae. A careful systematic survey by roentgenography, visualizing the many anatomic structures of the thorax, will usually explain and clarify the patient's clinical course.

INTRAVENOUS ANESTHESIA

A CLINICAL STUDY OF NINETEEN HUNDRED CASES

B. M. CARRAWAY, M.D. AND C. N. CARRAWAY, M.D., F.A.C.S.

Associate Surgeon, Norwood Hospital

Chief Surgeon and Director, Norwood Hospital

BIRMINGHAM, ALABAMA

THIS is a report of the experience gained in 1900 cases in which evipal and sodium pentothal were used as intravenous anesthesia at the Norwood Hospital during the past fifteen months. This has been found to be the most satisfactory anesthesia for all types of cases. This report will include 178 cases in which evipal was used and 1722 cases in which sodium pentothal was the anesthetic. Evipal was used at first, before sodium pentothal was placed on the market. Sodium pentothal, which has an added sulphur radical, gives a much smoother, more prolonged and less toxic anesthesia.

The preliminary medication is very essential in giving this type of anesthetic. Among the present group, 1600 received morphine and atropine before the operation and of these 25 per cent suffered from nausea or vomiting. Two-hundred received pantopon and of these only 10 per cent were similarly effected. For minor operations of short duration only atropine was given and 1 per cent of the patients were nauseated or vomited. Six received 4 gr. of sodium luminal early in the morning and atropine on call to the operating room, and were given morphine after reacting for pain. In these cases nausea and vomiting resulted in 50 per cent. A preliminary medication of an opiate and atropine is of definite value in obtaining satisfactory relaxation and preventing the formation of mucus in the air passages.

Before the anesthetic is administered, the patient is placed on the operating table, the field of operation is prepared and the patient properly draped. One of the arms is left out for the administration of the anesthetic. This procedure lessens the time in the operating room by about 50 per cent.

Intravenous anesthetics should be administered absolutely on the same principle as inhalation anesthetics and not according to the patient's weight, age or size. They should be given only by experienced anesthetists and according to the fractional method.*

Sodium pentothal and evipal are prepared in powdered form in vials of 1 Gm. Evipal is diluted with triple distilled water to 10 c.c. to make a 10 per cent solution; sodium pentothal is diluted to 20 c.c. to make a 5 per cent solution. Either solution is placed in a 20 c.c. syringe and is given very slowly while the patient counts out loud, also slowly. A competent nurse should stand at the patient's head while the anesthetic is administered. About thirty seconds from the time the anesthetist starts injecting the solution, the patient stops counting. At this time there is complete loss of sensation and reflexes. The jaw tends to drop and the tongue falls backward. Therefore, it is necessary for the nurse to hold the jaw up, and in a number of cases, to place an air-way in the mouth. At this stage the patient's skin is pallid, but the blood is always bright red; we have noted no ill effects. The needle is left in the vein throughout the operation; so that if the patient should move or groan at any time, an additional 1 or 2 c.c. of the solution may be injected. We have found, however, that there is no evisceration or

* Miss Anne Beddow, head anesthetist at Norwood Hospital, administered or supervised the anesthesia in all the cases here reported.

interference with abdominal relaxation, should this occur.

In the 178 cases in which evipal was used, there seemed to be a toxic effect in 60 per cent, causing twitching and spasm of the muscles of the extremities. There was a drop in blood pressure of from 10 to 30 points in 40 per cent. The respiration in most of the cases was decreased only 2 to 6 per minute and the pulse was increased 5 to 20. The reacting time varied, due to the amount of solution given. Fifty per cent of the patients were very excitable and irrational for about an hour while reacting. A number of patients had to be held in bed by the nurse.

Sodium pentothal gives a much smoother and more prolonged anesthesia than evipal. There is no tonic or clonic contraction of the muscles, but in 0.5 per cent of the cases there were slight cyanosis and hiccough, due apparently to sensitivity to the drug. There was relatively no change in the pulse or respiration; a drop of only 6 to 10 points in blood pressure occurred in 6 per cent of the cases. The reacting time after the use of sodium pentothal is governed, as with evipal, by the amount given. Usually if the anesthetic is stopped about the time the surgeon begins closing the wound, the patient will react very quietly in from ten to forty minutes. If large doses are given over a long period of time, the reacting time is longer, but the patient will react to such stimuli as a pinch or a pin-prick. After being aroused in this manner for a few minutes, he will then continue sleeping for several hours. In twenty-five cases of major operations, no opiates were given after the operation because the patient slept through most of the pain. No patient who received sodium pentothal was irrational or restless when reacting.

Of 900 cases, 125 were nauseated or vomited, 50 complained of gas pains and 30 of distention. None of these complained of headaches or ill feelings after the anesthetic. The following table lists the different types of surgical cases in which intravenous anesthesia was used:

Operation Performed	No. of Cases
Alexander and appendectomy	4
Application of cast	7
Aspiration of knee	1
Alexander	10
Alexander and perineorrhaphy	3
Amputation of extremities	53
Appendectomy	400
Albee's bone graft of back	1
Biopsy	3
Bottle operation for hydrocele	15
Bone graft in neck of femur	14
Bronchoscopy	2
Cesarean section	9
Cauterization of cervix	1
Cholecystostomy	3
Cholecystectomy	33
Cholecystectomy and appendectomy	9
Circumcision	12
Colostomy	5
Craneoplasty	5
Cryptorchidoplastic	1
Cystoscopy	33
Cystoscopy and fulguration	1
Dilatation and curettage	151
Diverticulectomy and appendectomy	1
Dressings	6
Encephalogram	1
Epididymotomy	8
Excision of hyperglossal cyst	5
Excision of tumor from gum	2
Exploratory laparotomy	18
Extraction of teeth	33
Eye operation	11
Fistula in ano	25
Fixation of ureter to psoas muscle	3
Forcep delivery	2
Freeing of post-operative adhesions	2
Gastric resection	10
Hemorrhoidectomy	60
Herniorrhaphy	94
Herniorrhaphy (bilateral)	15
Herniorrhaphy and appendectomy	9
Hysterectomy	44
Hysterectomy and appendectomy	62
Hysterectomy, cholecystostomy and appendectomy	2
Hysterectomy, appendectomy and perineorrhaphy	2
Hysterectomy and herniorrhaphy	1
Hysterectomy and salpingectomy	24
Incision and drainage	70
Insertion of Steinmann's pin	3
Intestinal anastomosis and appendectomy	1
Laminectomy	2
Ligation of tubes and appendectomy	9
Ligation of tubes and perineorrhaphy	3
Ligation of tubes, appendectomy and perineorrhaphy	4
Lumbar sympathetic ganglionectomy	2
Mastoidectomy	5
Mammectomy (simple)	11
Mammectomy (radical)	14
Nephropexy	11
Nephropexy and appendectomy	1
Nephrotomy	10

Operation Performed	No. of Cases
Nephrectomy	6
Orchidectomy	5
Perineorrhaphy	41
Plastic operation on face and extremities	12
Posterior gastroenterostomy	7
Removal of ingrowing toe-nail	2
Reduction of fracture	81
Reduction of fracture (open)	34
Removal of foreign body	11
Removal of hygroma	1
Removal of ovarian cyst	22
Removal of ovarian cyst and appendectomy	9
Removal of pilonidal cyst	3
Resection of presacral sympathetic plexus	1
Removal of semilunar cartilage	10
Removal of spur from heel	2
Removal of tumor from skin	29
Resection of peptic ulcer	14
Resection of peptic ulcer and appendectomy	1
Resection of rectum	3
Resection of prostate	9
Sequestrectomy	7
Suturing of lacerations and tendons	51
Salpingectomy	19
Salpingectomy and appendectomy	37
Shortening of round ligaments and appendectomy	16
Shortening of round ligaments and perineorraphy	1
Suprapubic	9
Skin graft	5
Trachelorrhaphy and perineorrhaphy	13
Thoracostomy	9
Tonsillectomy	12
Thyroidectomy	22
Urethrotomy (external)	7
Urethrotomy (internal)	5
Urethral dilatation	6
Vaginal hysterectomy	1
Varicocelectomy and appendectomy	1
Vasectomy	2
Wiring of jaw and extremities	11
Wiring of clavicle	1
Total	1900

The average time per operation in these 1900 cases was twenty-four minutes; the average amount given was 15 c.c.; and the average reacting time was one hour and thirty-four minutes. Intravenous anesthesia, especially sodium pentothal, is indicated for all types of surgical cases. So far we have found no contraindications.

In our series, the age distribution was as shown at top of column. The oldest patient was 88 years of age and the youngest 2 years. We did not use intravenous anesthesia in children under 2 years of age because of the difficulty of finding the veins.

Age	No. of Cases
2	5
3	5
4	17
5–10	67
10–20	334
20–30	657
30–40	431
40–50	224
50–60	90
60–70	36
70–75	10
75–80	9
over 80	15

The longest operation was a prostatic resection in a patient 78 years of age, which took two hours fifty-five minutes. This was the time for the actual operation and control of hemorrhage. The largest amount of anesthetic given was 68 c.c.; this was administered to a patient 38 years of age for a herniorrhaphy. The smallest amount used was 2 c.c., in a patient with a ruptured ectopic pregnancy who was on the borderline of shock. This operation lasted twenty minutes. On one occasion operation was performed on a patient with a toxic thyroid, who was very nervous. In this case the anesthetic was started in the patient's room and she was immediately rolled to the operating room where a thyroidectomy was done. This is not advised as a routine procedure, but it does very nicely in selected cases. The anesthetic can be given repeatedly to an individual case, as was done on eleven different occasions within a period of three months without any ill effects. It may also be given in cardiac disease and hypertension. One of our patients had a blood pressure of 260/140, hysterectomy and appendectomy were done. Two bronchoscopies were performed and lung abscesses opened into the bronchus. Although the purulent material was coughed up, there was perfect relaxation for the passing of the bronchoscope. Sodium pentothal is so pleasant that a large number of patients with compound fractures, burns, etc., who had had it on admission, have asked for it merely to have dressings changed.

So far, there have been no fatalities due to the anesthetic and no post-operative

complications. The only complications during anesthesia have been cyanosis, hiccoughs, coughing and occasional blockage of the air passage with mucus. For this, sodium benzoate, alpha lobelin, picrotoxin, oxygen and carbon dioxide. Only eight cases, or .05 per cent had to receive coramine and amyl nitrite for cyanosis.

INTRAVENOUS ANESTHETIC RECORD

Case No. Room 227 Date September 28, 1937

Patient's Name Mr. George Rylant Age 38

Pre-operative Diagnosis Ruptured Peptic Ulcer

Post-operative Diagnosis Ruptured Peptic Ulcer

General Condition of Patient Grave—Calcium Chloride Grs IV Intravenously just before operation.

Pre-operative Blood Pressure Systolic 120 Diastolic 82

Temperature 98 Respiration 6 Pulse 104

Pre-operative Drugs Administered Atropine Gr $\frac{1}{150}$

Surgeon Dr. C. N. Carraway

Anesthetist A. Beddow

Anesthetic Sodium Pentothal, XI c.c. 5% Intravenously

Condition During Anesthesia Respiration Labored and Irregular from Morph., 8 to 16
Pulse, Vol. Good 78–94 B.P. 156/100

Operation

Time Began 9:50 p.m. Closed 10:35 p.m.

Condition Just After Operation Very Good Pulse 100 Resp. 20

Time Reacted 12:10 a.m. Blood Pressure Sys. 148 Dias. 84

Nauseated No length of time

Vomiting None length of time

Gas Pains None length of time

Distention None length of time

Remarks: Patient was given Morph. Gr $\frac{\ddot{s}\ddot{s}}{}$ before admission.
Patient reacted quietly and has not complained of any headache.
Not restless.

stimulants are given, such as 5 c.c. of coramine intravenously and amyl nitrite as an inhalant. Other cardiorespiratory stimulants may be used, such as caffeine

An electrocardiographic study was made in ten major operations, the first the night before the operation, the second during the operation and the third ten hours after the

operation. The tracings did not reveal any disturbance in the rhythm or rate and did not show any myocardial damage.

Post-mortem examinations were made on twelve patients who had fully recovered from the intravenous anesthetic, but who died several days after the operation from other causes. A careful microscopic examination did not reveal any changes in the organs due to the anesthetic.

Sodium pentothal was used in a large number of cases of experimental animal surgery. At first it was given according to the dog's weight, but one animal expired from an overdose. The respiration was affected first; the heart did not stop for several minutes. In the rest of the cases it was administered by the fractional or intermittent method and was very successful.

In gathering these data on intravenous anesthesia, a special anesthetic record sheet was placed on each surgical chart and information recorded. (See Hospital Chart.)

SUMMARY

1. Intravenous anesthetics should be administered only by experienced anesthetists with the assistance of competent nurses.

2. They should be given only by the fractional method.

3. A preliminary sedative is essential. Pantopon and atropine cause less nausea and vomiting than other drugs.

4. Sodium pentothal may be used in any type of case and over a long period of time with perfect relaxation.

5. There is no fall in blood pressure or myocardial damage.

6. Sodium pentothal gives a longer, smoother and less toxic anesthesia than evipal.

7. The respiratory centers are affected before the cardiac centers.

ANALGESIA IN LABOR: ONE YEAR'S EXPERIENCE WITH THE NEW ANALGESIC SIGMODAL

FREDERICK V. EMMERT, M.D. AND SIEGFRIED GOLDSCHMIDT, M.D.

ST. LOUIS, MISSOURI

ALMOST two years ago, we introduced a new method of alleviation of pains in labor in a discussion[1] of the symposium on twilight sleep before the American Medical Association in Kansas City. Some time later, we published a preliminary report[2] concerning our experiences with the new barbiturate, sigmodal, and gave our method and results in a detailed report of forty-five cases. In the meantime, we continued our work and we feel justified in drawing definite conclusions, reviewing our experiences of the past year.

As pointed out in our first paper, the trend in obstetrical analgesia is away from the morphine-hyoscine semi-narcosis and toward the increased use of barbiturates. The voluminous literature covering this subject shows that barbiturates produce satisfactory analgesic effect in labor without undue danger to the baby.

In contrast to the usual oral administration of barbiturates, sigmodal is given by rectal application. Its chemical formula is sodium-amyl-beta-bromallyl-malonylurea. Sigmodal is marketed in a 10 per cent solution, stabilized in 10 per cent antipyrine. It has no irritating effect on the rectal mucosa and is absorbed rapidly. Its prompt hypnotic action is due to the presence of an asymmetric C-atom. The drug is decomposed so quickly in the blood stream that only traces appear in the urine. We have reason to assume that sigmodal is transmitted to the fetus in such small amounts, if at all, that it does not interfere with the vital activities. This may explain why we did not observe any deleterious or obnoxious effect upon the infant before or after delivery.

We desire to repeat our method of procedure. Particular cases were not selected, but we used the drug routinely. Due to the rapid analgesic effect, we were able to obtain very satisfactory results, even in multiparae in the late stage of labor. Patients with toxemias were not excluded and the drug proved its usefulness in these complications. Hemorrhoids and fistula-in-ano do not interfere with the use of the drug.

We wish to stress the statement from our previous report that an *absolute* requirement for the time of administration is that the patient is definitely in labor and that contractions occur regularly and effectively. False labor and too early administration of the drug prolong the delivery unduly and deprive the patient and the doctor of the advantages of the procedure. A second requisite is an empty bowel. It is quite evident that large amounts of feces, occupying the lower rectum, render the absorption of the drug difficult and eliminate part of its effect. Therefore, a cleansing enema should be given in the beginning of labor.

The time of administration of the drug is important if the full effect is to be obtained. Our experiences taught us that sigmodal may be administered with most benefit to the patient if the dilatation has progressed to two and one-half fingers in primparae or one finger in multiparae, provided labor is established.

The application of sigmodal is done rectally. The patient is placed on her left side and a urethral rubber catheter, which fits on a 10 c.c. luer syringe, well lubricated, is inserted into the rectum as high as possible, preferably above the fetal head. It is an advantage to choose for the moment of introduction the interval between pains, and inject slowly through the catheter 10 c.c. of the drug, followed by 5 c.c. of saline, in order to assure the full

dose. After removal of the catheter, pressure is exerted on the anus for several minutes by means of a pad. Ten cubic centimeters of sigmodal have proved to be without any deleterious effect on mother or child, and much larger doses have been given during our experimental stage without untoward results.

The action of the drug becomes effective within fifteen minutes, and increases for at least two hours, when a gradual decrease in intensity sets in. The duration of the analgesia has been from five up to more than ten hours. In several cases, where an unexpectedly long labor was encountered, the administration of the drug in amounts of 5 c.c. has been repeated, keeping the patient under analgesia for several additional hours. The first effect of the drug upon the parturient woman consists in producing drowsiness. Gradually the patient falls into sleep which is quiet in most cases throughout labor. We encountered no unwarranted effect on the mother except in one case, where twitchings occurred and the depth of sleep was unusual. This patient evidently had a hypersensitivity to barbiturates; 2 c.c. of coramine was sufficient to restore the normal response without harmful effects. Normally, the sleep of the patients is not deep enough to exclude their coöperation, which acts favorably upon the normal mechanism of labor.

After delivery, our patients enjoyed a sound after-sleep for several hours, which we consider beneficial for its contribution to the rapid recovery of physical and mental functions. The above-mentioned patient showed a prolonged after-sleep which, however, easily could have been interrupted with a few c.c. of coramine, if desired.

Our former favorable observations in respect to restlessness were confirmed by the experiences in our last series. The majority of our patients slept peacefully throughout labor. Only a small minority reacted to contractions with reflex movements or slight moaning and groaning, falling back into sleep between contractions. Remarkable restlessness occurred in only four of our series. There was no comparison with the amount of restlessness and excitement which we find so often after the use of morphine- or nembutal-hyoscine semi-narcosis.

In a few cases where the initial contractions before the administration of sigmodal were felt to be unusually painful due to hypersensitiveness of the patient, we overcame this interval by the subcutaneous injection of $\frac{1}{48}$ gr. of dilaudid. This drug is said to interfere less with contractions than morphine.

The loss of blood in the third stage of labor was normal or very moderate in our second series, except in two cases in which post-partum hemorrhages reaching 800 c.c. occurred. In both cases the bleeding was controlled by the intravenous medication of 3 minims of pituitrin with immediate effect and without any disadvantage to the mother.

We encountered the following complications: one case of severe toxemia and one of mild degree; five breeches and five occiput posterior positions. Two of the breech presentations were born spontaneously. In two of the occiput posterior positions, labor was difficult and prolonged.

As we have previously stated, all our babies were born in good condition and cried at once. No interference with spontaneous respiration was noted and no resuscitation was required.

Of our second series of eighty cases, thirty came from associates who kept accurate data. As a matter of precaution, they used in their first cases only 6 to 8 c.c. of the drug. This smaller dosage decreased the percentage of total analgesia in their first observations; however, they attained our figures as soon as they accepted our original dosage of the drug.

We give the figures of our first report, pertaining to forty-five cases, thirty of which were primiparae, and of the present paper which embraces eighty cases, fifty-eight of them primiparae. Finally, we

First publication (45 cases):

	Complete, Per Cent	Partial, Per Cent	None, Per Cent
Primiparae:			
Analgesia	80	13.3	6.7
Amnesia	80	13.3	6.7
Multiparae:			
Analgesia	73.3	20	6.7
Amnesia	73.3	20	6.7

Average duration of labor:
Primiparae: 13 hours, 43 minutes
Multiparae: 11 hours, 4 minutes
Average duration of medication:
Primiparae: 3 hours, 57 minutes
Multiparae: 3 hours, 42 minutes

Present series (80 cases):

	Complete, Per Cent	Partial, Per Cent	None, Per Cent
Primiparae:			
Analgesia	67.24	30.68	2.08
Amnesia	74.14	24.14	1.72
Multiparae:			
Analgesia	72.7	22.7	4.6
Amnesia	81.8	13.6	4.6

Average duration of labor:
Primiparae: 15 hours, 37 minutes
Multiparae: 10 hours, 27 minutes
Average duration of medication:
Primiparae: 4 hours, 26 minutes
Multiparae: 3 hours, 1 minute

Total of two series (125 cases):

	Complete, Per Cent	Partial, Per Cent	None, Per Cent
Primiparae:			
Analgesia	71.59	25	3.41
Amnesia	76.13	20.45	3.42
Multiparae:			
Analgesia	73	21.6	5.4
Amnesia	78.4	16.2	5.4

Average duration of labor:
Primiparae: 14 hours, 58 minutes
Multiparae: 10 hours, 42 minutes
Average duration of medication:
Primiparae: 4 hours, 16 minutes
Multiparae: 3 hours, 17 minutes

shall summarize the total, consisting of eighty-eight primiparae and thirty-seven multiparae.

As is shown by the above figures from 125 consecutive confinements, the total time of labor in primiparae was fourteen hours and fifty-eight minutes, and in multiparae, ten hours and forty-two minutes. The duration of medication in primiparae was four hours and sixteen minutes and in multiparae, three hours and seventeen minutes. These figures demonstrate clearly that the duration of labor after the use of sigmodal was not prolonged, but definitely shortened. The average length of medication time showed that the drug does not produce any delaying effect upon the force and frequency of contractions. The absence of maternal morbidity and mortality and the fact that no fetal deaths were encountered emphasizes the harmlessness of sigmodal to mother and child. The amount of restlessness after the use of sigmodal compares favorably with that of every other method of alleviation of pain in labor.

Thus, we feel entitled to confirm the conclusions drawn in our preliminary report:

1. Sigmodal can be administered without danger to mother or child.
2. It does not interfere with labor or the effects of contractions.
3. It shortens the duration of labor in most of the cases.
4. Complete amnesia occurs in nearly 80 per cent of the cases.
5. Sigmodal is a safe drug in the hospital and in the hands of the general practitioner in the home.
6. Finally, it approaches more nearly the ideal analgesia in obstetrics.

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MANAGEMENT OF CALCULI IN THE LOWER THIRD OF THE URETER

ARBOR D. MUNGER, M.D., F.A.C.S.

Attending Genitourinary Surgeon and Chief of Section, Lincoln General Hospital

LINCOLN, NEBRASKA

TO all alike does not come the gift of simplicity in the solution of a presented problem. Especially is this manifest in the art of technical application throughout the science of surgery. Among certain gifted urologists the problem of management of calculi in the ureter seems to have been simply solved, while to others the same problem is still surrounded with considerable complexity.

Aretaeus stated, "Nature did well in forming the cavity of the kidneys oblong and of equal size with the ureters and even a little larger, so that if a stone formed above, it might have ready passage to the bladder." However apt the applicability of this utterance might have been at the time it was recorded, the half truth of the statement at the present time is established through the evolution of our more refined diagnostic methods. To Aretaeus, the stones which passed were all-important; those which nestled in some part of the ureter, he had not the armamentarium to discover.

Of the stones which do not pass, most will be found in the ureter below the level of the rim of the bony pelvis. Statistics reveal that ureteral stones are found in this location in about 60 per cent of cases, with a selective preponderance for the ureterovesical junction.

When a patient presents himself with a stone lodged in the lower third of the ureter, the question arises as to what procedure should be instituted for its removal. Should the patient be subjected to dilatation or repeated dilatations, and, if so, over what period of time should such dilatations be continued? Should open operation be instituted to the exclusion of other procedures? Are there diagnostic criteria which can be invoked to determine the course of procedure? If not, what factors in the patient's picture provide a basis for judgment? A comparative study of reports from urologists throughout the country with respect to management of this condition has been very illuminating.

Manipulative removal is successful in from 50 per cent to 98 per cent of cases, depending upon the urologists reporting. This wide range is among equally good urologists. Why this divergence in end results? Are some more skilful at endovesical manipulation than others? Only occasionally is this so. I have a profound respect for the urologist who can deliver 70 per cent or more of calculi from the ureter by manipulative means, but I feel unable to align myself with them. My judgment prompts me to do an operation early in a certain percentage of cases, since I feel that the kidney is spared by careful operation far more than if a long drawn-out, conservative plan of procedure is adopted. Although conservative measures may be successful in getting out the stone, the delay gives the kidney time to go on to partial destruction. Peacock reports 50 per cent passage of calculi by manipulative means, Squires and Crowell 87 per cent, Dourmaskin 70 per cent, Walther 88.9 per cent, Ballenger and Elder 90 per cent, Barney and Chute 51.5 per cent, Ravich 85 per cent, and Mathe 78.3 per cent. Koll in a discussion of Barney's paper, states that in a series of 200 cases, 98 to 99 per cent either were passed spontaneously or were removed by endovesical procedures. Koll further states, "To cut down and dissect out a ureter for the removal of a ureteral calculus is subjecting the patient, in the vast majority of instances, to a

subsequent stricture of the ureter which will later necessitate nephrectomy." If this be true, it is a grave indictment of a great number of urologists who institute open operation for the removal of ureteral calculi in from 30 to 60 per cent of cases.

In this review of 138 proved cases of calculi in the lower ureter occurring over a period of twelve years, there were ninety-eight males and forty females. The age incidence ranged between 21 and 80 years with an average incidence of 43.1 years. Seventy-one had calculi on the right, sixty-six were on the left side, and one case was bilateral. All cases in this series, except eight, were subjected to from one to five endovesical manipulations. Of the eight cases not subjected to cystoscopy, seven, or 5.0+ per cent, passed their calculi voluntarily and either refused cystoscopy or were dismissed without it.

The remaining case in which cystoscopy was not done was an instance of bilateral calculi in a woman 39 years of age. There was a definite history of acute impaction on the right side one year previous, for which an emergency appendectomy was done by a general surgeon, and the present admission was for a similar acute syndrome on the left side. When seen about sixty hours after onset there was but very little sanguino-purulent urine in the bladder, the temperature was elevated, and the tongue dry. *X*-ray revealed an almond seed-shaped calculus in the lower end of each ureter. The patient was desperately sick and it was felt in this instance that cystoscopy would be a case of "fiddling while Rome burned." Hence an emergency ureterolithotomy was performed on the left side. Local recovery was prompt with rapid return of function. The patient, however, continued to run a very septic course, and herein lay the error in judgment: she should have had a ureteral catheter if possible implanted into the right ureter at the time of her first operation. Ten days later the calculus was removed surgically from the right ureter and a nephrostomy done for a massively infected pyelo-ureterectasis. This did not suffice, however, to save the kidney, and eventually nephrectomy was performed. Recovery was uneventful thereafter with no subsequent trouble in the left ureter for over a period of ten years.

In sixty-five, or 47.1 per cent of cases, calculi were passed after one to five endovesical manipulations. In twenty-one of these sixty-five cases, one cystoscopic procedure alone caused the passage of the calculus. Unquestionably in some of these cases passage of the calculus would have occurred without cystoscopic procedure. Nevertheless, cystoscopy was instituted, the calculus manipulated, and credit given for at least facilitating its passage. In two cases the calculus was protruding from the ureteral orifice and mere manipulation with a catheter completed the passage.

In forty-four manipulation cases, two or more cystoscopic procedures were instituted. It is interesting to note that cases which came under care during the first four or five years of this report were pertinaciously manipulated for the removal of calculi; whereas later cases show decidedly less of this tendency and are more in favor of earlier operative interference. With this leveling-out process has come a decidedly more balanced urologic sense. An attempt to analyze the reasons for this change is most interesting. During the first few years in urologic practice, in the period in which he is gaining a foothold, the urologist is apt to become the victim of his medical confrères. He has a feeling that it is necessary to justify his existence as a urologist. Eventually he sees a patient with a calculus impacted in the ureter. The family physician knows that his tried friend, the general surgeon, can relieve the situation by operating, but the urologist has talked about removing calculi without operation, and the patient and family readily accept this desirable alternative. The family physician therefore makes a blunt request for the removal of the calculus without operation. The urologist's judgment becomes stultified, and he dilates

and prays, and prays and dilates. Fortunately, in a goodly proportion of cases his effort is rewarded, but unfortunately some patients tire of the so-called experiment and disappear in dissatisfaction, to be operated on elsewhere. Yet as time goes on and the urologist's place is more firmly secure, his own judgment supersedes, and he himself dictates the procedure.

It is not an unusual thing for patients under multiple dilatations to come to fear the procedure more than an operation. In not a few instances, post-manipulative reactions have been far more severe than post-operative upsets. Temperature reactions with chills are occasionally extremely vicious and prolonged. Frequently there have been several days of progressive oliguria with marked urosepsis and elevation of blood non-protein nitrogen. Practically all types of stone dislodgers and ureteral instruments have been used with no universally brilliant results. In one case in which I used a new spiral-tipped dislodger, the spiral tip with its little ball on the end separated and was left in the ureter gently reposing above the calculus. Meatal enlargement, where possible with fulguration, and catheter dilatation, with single or multiple catheters left in situ give the best results.

Just how much ureteral manipulation should be persisted in before a decision can be made that further manipulation is improvident, becomes a question of paramount importance. Certainly there is a tendency to persist in manipulation beyond good reason. It will prove a wise course to approach each case with the opinion that, if the calculus does not move after two or three thorough attempts, it is probable that the calculus is fixed and should be removed surgically. While adherence to this program results in more surgical removals, yet a concretely defined and expeditiously instituted procedure will bring satisfactory end results and satisfied patients. Let me quote the Editor's footnote from a review of compiled articles on this subject in the *Year Book of Urology*, 1935; "It is unquestionable that urologists are persisting too long in attempts to cause ureteral stones to pass by cystoscopic manipulation. Such conservatism causes more physical discomfort than surgical removal, and in fact, it is usually found that stones not dislodged by the usual cystoscopic methods, are embedded, by spicules, in the ureteral walls. It is very poor practice to persist in non-operative procedures if the stone has not made progress after two or three cystoscopic manipulations."

Sixty-six, or 47.8 per cent, of the 138 cases in this series were subjected to ureterolithotomy; 1 bilateral, 41 right, and 21 of the left ureter. Thirty-eight of these cases were operated on as manipulative failures, after from two to four attempts had been made to recover the stone. In the remaining twenty-eight cases there was but one cystoscopic procedure, and where possible an indwelling ureteral catheter was implanted with a coincident decision to remove the calculus surgically. A study of the contraindications to continued manipulative procedures reveals that in most instances there was an infection above the impaction of sufficient magnitude to cause a severe systemic reaction, and making catheterization impossible or extremely difficult. If the calculus impaction is of some duration, and there is an acute reaction, operation is necessary to preserve renal integrity. The size of the stone has less influence on the decision to operate than the duration and severity of impaction and the intensity of the general picture.

A calculus in the ureter does not confine its pathologic changes to the immediate tissue involved. It does irreparable damage to the kidney, and in infected cases is a potential hazard to the entire physical economy. The chronically infected calculus, with long standing round-celled infiltration of the entire ureteral wall, with its attendant thickening and rigidity, with marked periureteritis, make extreme caution necessary in endovesical manipulation. Such

ureters are especially susceptible to rupture, and in a certain number the calculus will have already perforated the ureter.

In reviewing the reports by certain urologists of the large numbers of calculi removed by manipulation, one is impressed with the paucity of infection and its troublesome sequelae. On the other hand, approximately 35 per cent of the cases in this small series have had infection of sufficient virulence to cause hesitancy in dilating beyond the simple implantation of a ureteral catheter for drainage. To traumatize and massage with dilating instruments such a badly infected field is surgically unsound. Such patients, in my hands, have not done at all well under such a régime.

There have been two deaths in this series. The first followed an operation for removal of a calculus in the right lower ureter impacted just above the bladder in a man 64 years of age. The symptoms had been of but a few hours' duration and the impaction impassable; chills and high temperature were present. At operation, the ureter was angry and greatly enlarged, the lower 3 or 4 cm. markedly thickened with dense periureteritis. The calculus, 1 by ½ cm., was in a sacculation and completely obstructed the lumen. There were syrupy, foul-smelling, purulent ureteral contents with a pyonephrosis. Nephrostomy was instituted but a progressive urosepsis and death ensued. Autopsy revealed both kidneys with multiple hematogenous abscesses.

The second death occurred early in this series, following repeated ureteral dilatations with subsequent passage of a not too small calculus from the right ureter. Progressive sepsis with anuria, general peritonitis and death followed. Culture from the urine and peritoneal secretion grew a short chain streptococcus. Autopsy presented an infected pyelo-ureterectasis with both kidneys showing an acute pyelonephritis. This patient would probably have had a better chance to avoid his peritonitis had he not been stirred up by repeated manipulations. This case in itself decidedly enhanced my respect for the dangers of repeated ureteral dilatations in the presence of infection.

One operative case presented the interesting complication of acute ureteral leakage with retroperitoneal extravasation four weeks after the patient was discharged from the hospital, primarily healed. This occurred while the patient was lifting some farm machinery. Incision and drainage to the ureter with a large indwelling ureteral catheter corrected the situation without further trouble. After surgical removal of the calculus, this patient's lower ureter should have been dilated. There must have been sufficient ureteral obstruction with hydrostatic pressure to cause the inadequately healed ureteral incision to give way. Dilatation of the ureter before the discharge of the patient from the hospital is now a routine measure.

Two incisions have been used, the first, a median suprapubic incision as described by Judd; the second, an oblique lower quadrant muscle-splitting incision as described by Gibson. The median incision has nothing to commend it, unless perhaps one should desire to remove bilateral calculi in one operation. The ureter is difficult to locate and exposure not entirely adequate, whereas, with the oblique incision there is far greater exposure, with much greater ease in locating and isolating the ureter. Contrary to Barney's experience, I have not found it difficult through this incision to expose the juxtavesical portion of the ureter. In fact, it is much easier than through the median incision. In the male the vas deferens and the obliterated hypogastric artery serve as guides to the ureter; in the female the round ligament leads to the broad ligament and serves as a retractor. In difficult cases a lateral cystotomy has been made, thus combining a mixed trans- and laterovesical approach. Hemostasis is a negligible factor in both incisions, and both are extraperitoneal. It is not necessary to suture the ureter. The ureter is always explored upward and

downward with a ureteral catheter. In the occasional case where there is an inordinate amount of periureteritis with coincident trauma at time of removal of the calculus, healing has been more or less delayed with urinary leakage for an undue period. The early removal of drain from the operative site will facilitate ureteral healing.

For such cases an indwelling ureteral catheter has been inserted through a counter-incision higher in the ureter away from the thickened area. Such procedure has facilitated irrigation of a badly infected ureter and kidney pelvis, with the further advantage of keeping the ureteral operative site dry, thus promoting quick healing. More recently a good-sized indwelling catheter has been implanted in the ureter following operation with almost spectacular healing results.

I should like to take exception to the view that the operative removal of ureteral calculi subjects the patient in a vast majority of instances to a subsequent stricture of the ureter, which will later necessitate nephrectomy. An occasional kidney will have to be removed in any series of impacted calculi, but certainly it is not the result of the operation; it is probably the result of lack of operation in the presence of a severe infection.

In conclusion, it may be said that the inadequacy of this review and the small series of cases are appreciated. The cases have, however, served to establish a definite viewpoint on procedure. The operative incidence has been greater than shown in most reports.

Lastly, let it be emphasized that the mere removal of a calculus is not the paramount consideration; rather, if we are fair to our patient, this is only the initial step along the pathway of recovery. A minute study of all the avenues of calculi production, with attempted correction of the same, and follow-up maintenance of the integrity of the ureter, constitute the true function of the urologist. To do less than this detracts from the purposes of urology as a specialty.

REVIEW OF THE "V" TYPE NEPHROTOMY FOR STAGHORN CALCULUS WITH SPECIAL REFERENCE TO FINAL RESULTS*

GEORGE C. PRATHER

BOSTON, MASSACHUSETTS

THE immediate results following the "V" type of nephrotomy described in 1934,[1] proved to be very satisfactory. an excellent exposure of the inside of the kidney for the removal of any additional stones. (Fig. 2.)

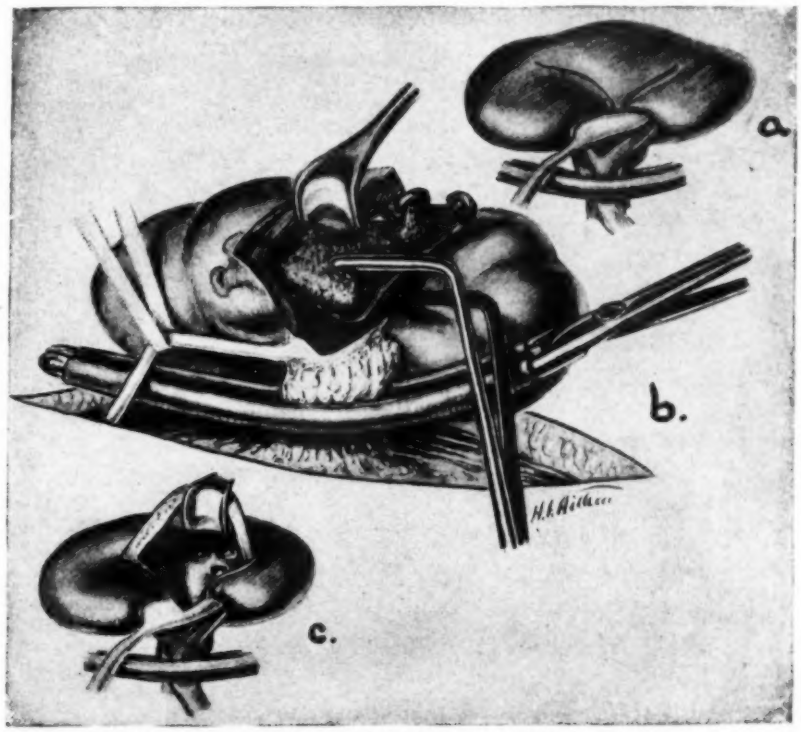


FIG. 1. Pedicle clamp in place (left kidney). A, "V" incision on posterior surface of kidney. B, removal of stag-horn calculus. C, flap retracted; visual access to all calices.

It seems wise, however, to review the condition of the patients, and particularly of the kidneys, after an interval of several years, to see if the longer term results justify such a procedure.

In the operation referred to, an intestinal clamp was placed on the renal pedicle for short periods of time in order that a "V" type incision might be made on the posterior surface of the kidney in a bloodless field. (Fig. 1.) This facilitated prompt removal of the staghorn calculus in one piece and gave

The data regarding the effect of short periods of anoxemia on renal tissue, the methods of suture of nephrotomy wounds, and the detailed case histories which were given in my paper of three years ago[1] need not be repeated.

The patients whose cases are to be discussed are those reported in 1934. Additional post-operative *x*-ray data have now been secured, making it possible to study their renal condition at a period approximately three to five years after operation.

* From the Urological Service of the Beth Israel Hospital, Boston, Mass.

The urine sediment in the case reports consists of the centrifuged specimen, examined microscopically with the 4 mm. objective and the 10 × ocular. Appearance time refers to the first appearance of color from the ureteral catheter following the intravenous injection of 1 c.c. of phenolsulfonphthalein. Kidney function is calculated from the urine specimen collected during the first fifteen minutes following the appearance of phenolsulfonphthalein through the ureteral catheter. The specimens are diluted to 500 c.c. and compared with colorimetric standards.

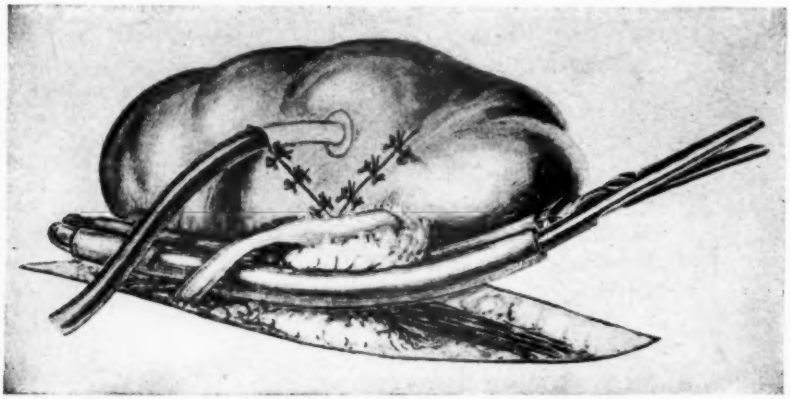
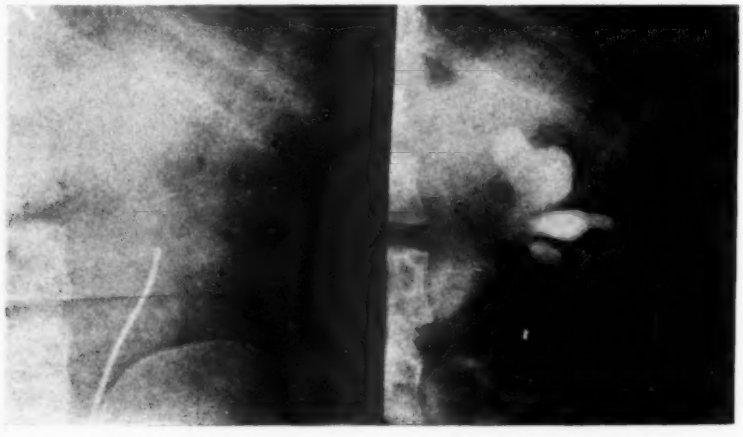


Fig. 2. Closure of nephrotomy incision (left kidney) with nephrotomy tube in place.

Case 1. I. K., age 30 years.

Operation. April 29, 1933, a left "V" type nephrolithotomy was done with hemostasis by intestinal clamp on the renal pedicle. The pedicle clamp was loosened every ten minutes. A staghorn calculus was removed, in addition to numerous small, hard stones from the lower calices.

Stone Analysis. Calcium oxylate, calcium and magnesium phosphate, triple phosphates.



A B

Fig. 3. Three years and nine months after operation. A, plain K. U. B. plate. B, retrograde pyelogram.

Pre-Operative Urine Report. Left kidney sediment; rare w.b.c. Left appearance time, five minutes. Left kidney function, 5 per cent.

Pre-Operative X-ray Data. A large branching calculus filled the entire left kidney pelvis and calices. Left pyelogram showed the stone filling the entire pelvis and lower calyx. The upper calyx was markedly dilated and irregular.

Post-Operative X-ray Data. Pyelogram seventeen days after operation showed numerous small calcified bodies in the lower pole

A B

Fig. 4. Case 11. Left kidney. A, pre-operative K. U. B. plate. B, pre-operative retrograde pyelogram.

A B

Fig. 5. Case 11. Left kidney. A, K. U. B. plate twenty-one days after operation. B, retrograde pyelogram twenty-one days after operation.

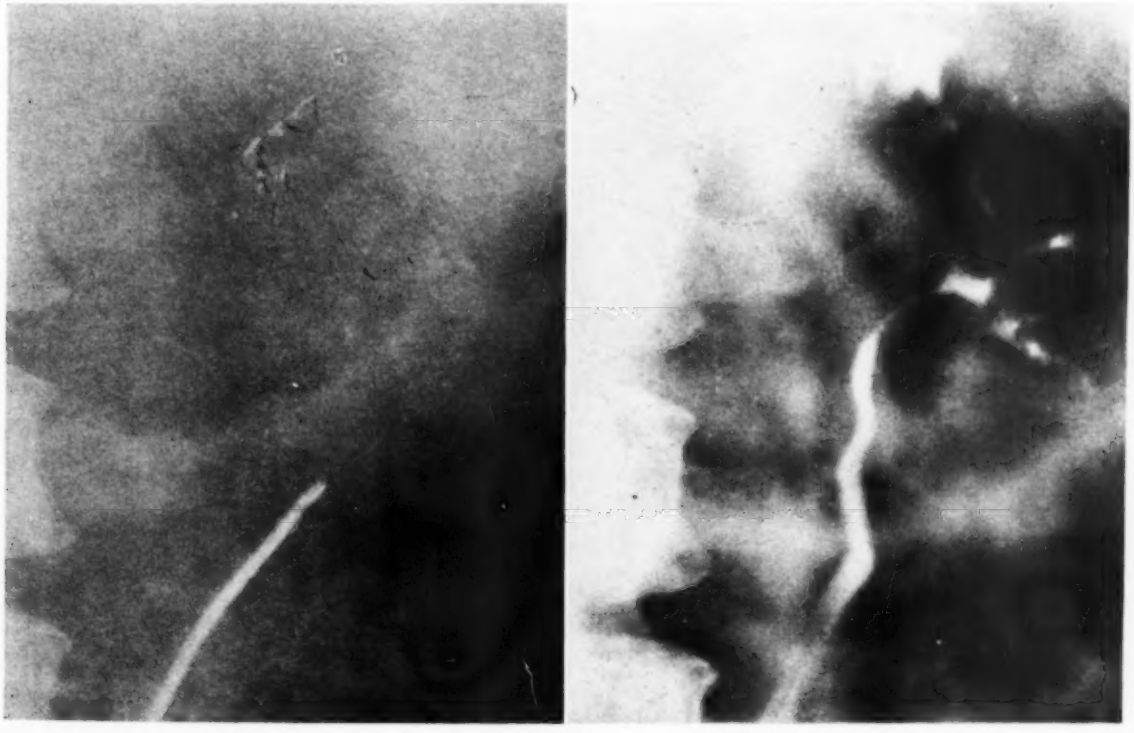
Fig. 6. Case 11. A, K. U. B. plate six months after operation. B, K. U. B. plate one year after operation. C, retrograde pyelogram one year after operation.

A B

Fig. 7. Case 11. Left kidney. A, K. U. B. plate two years after operation. B, K. U. B. plate before operation, showing both kidney and bladder stones.

A B

Fig. 8. Case III. Left kidney. A, pre-operative K. U. B. plate. B, pre-operative retrograde pyelogram.



A B

Fig. 9. Case III. Left kidney. A, K. U. B. plate seven weeks after operation. B, retrograde pyelogram seven weeks after operation.

of the left kidney. The pelvis was well outlined, slightly compressed. The upper and middle calices were dilated, the lower calices incompletely filled.

Late Post-Operative Urine Report. December 23, 1936, the left kidney showed sediment, 3–5 w.b.c., bacilli. The *pH* was 7. Appearance time: left, ten minutes. Left kidney function; 1 per cent (poor drainage).

Late Post-Operative X-ray Data. There were small discrete areas of calcification in region of the lower pole of the left kidney apparently in the kidney cortex. Left pyelogram showed dilatation of the calices. The pelvis was small, and the ureter not filled. (Fig. 3.)

Case 2. F. C., age 61 years.

Operation. March 28, 1934, a left "V" type nephrolithotomy was done with hemostasis by intestinal clamp on the renal pedicle. The pedicle clamp was loosened every eight minutes. A large stone, forming cast of the kidney, was removed, and small calculi were removed from various calices.

Stone Analysis. Calcium and magnesium phosphate, calcium oxylate.

Pre-Operative Urine Report. Left kidney sediment; 2–3 w.b.c. Left kidney culture B. coli. Left kidney function, 7 per cent.

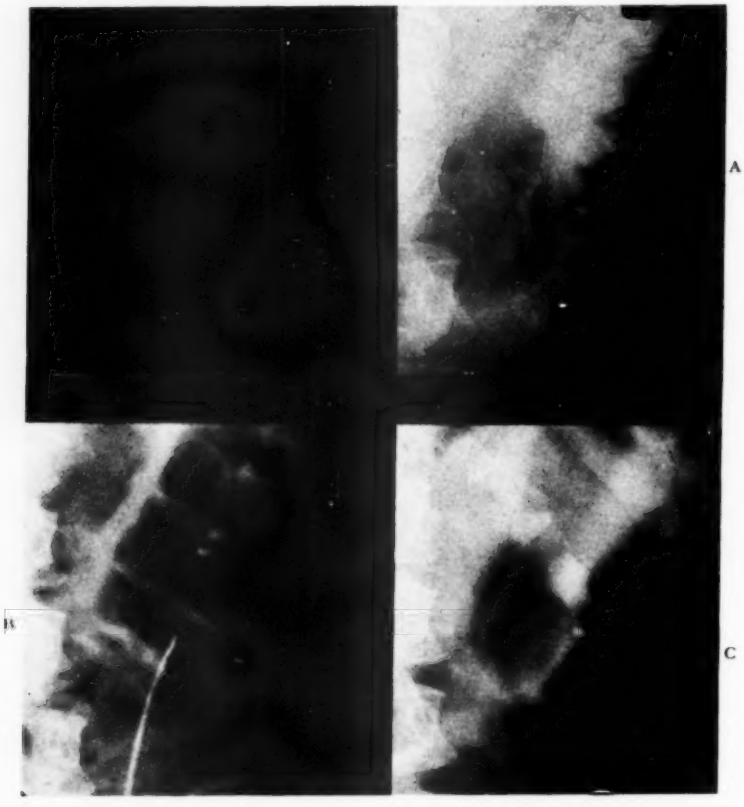


Fig. 10. Case III. Left kidney. A, intravenous pyelogram one year and four months after operation. B, plain K. U. B. plate three and one-half years after operation. C, retrograde pyelogram three and one-half years after operation (incomplete filling).

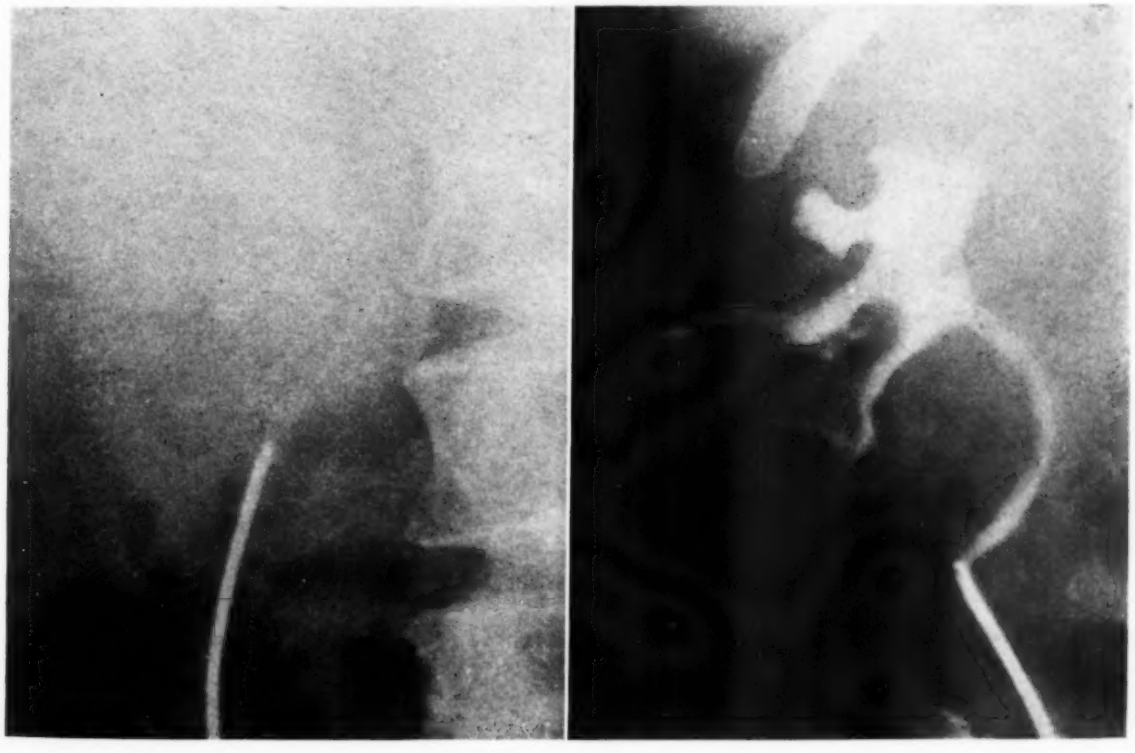
Pre-Operative X-ray Data. A large calculus filled the entire pelvis and calices of the left kidney. There was also a large stone in the bladder, measuring 4 by 6 cm. A left pyelogram showed a large stone filling the entire kidney.

Post-Operative Urine Reports. March 26, 1935, left kidney sediment; 8–10 w.b.c., B. proteus. Left appearance time, five minutes. Left kidney function, 1 per cent.

Post-*Operative X-ray Data.* Pyelogram twenty-one days after operation showed question of small area of calcification in region of kidney. The upper calyx was not dilated, and the lower calyx not filled. The renal pelvis was small. K.U.B. plate, September 27, 1934, showed an area of calcification filling the left renal pelvis. Pyelograms, March 26, 1935, showed the left renal pelvis filled with a large

A B

Fig. 11. Case III. Right kidney. A, pre-operative K. U. B. plate. B, pre-operative retrograde pyelogram.



A B

Fig. 12. Case III. Right kidney. A, K. U. B. plate sixteen weeks after operation. B, retrograde pyelogram, sixteen weeks after operation.

calcified body. The ureter was considerably dilated. K.U.B. plate, April 12, 1936, showed the calcified body in the region of the left pelvis still present. (Figs. 4, 5, 6 and 7.)

Case 3. I. C., age 43 years.

culture, B. proteus. Right appearance time five minutes. Right kidney function, 7 per cent. Left kidney sediment; 75 w.b.c. Left kidney culture, B. proteus. Left appearance time six minutes. Left kidney function, 5 per cent.

Fig. 13. Case III. Right kidney. A, intravenous pyelogram one year and three months after operation. B, K. U. B. plate three years and four months after operation. C, retrograde pyelogram three years and four months after operation.

First Operation. July 11, 1933, a left "V" type nephrolithotomy was done with hemostasis by intestinal clamp on the renal pedicle. The pedicle clamp was loosened every eight minutes. The stones were removed and the calices washed with boric acid solution.

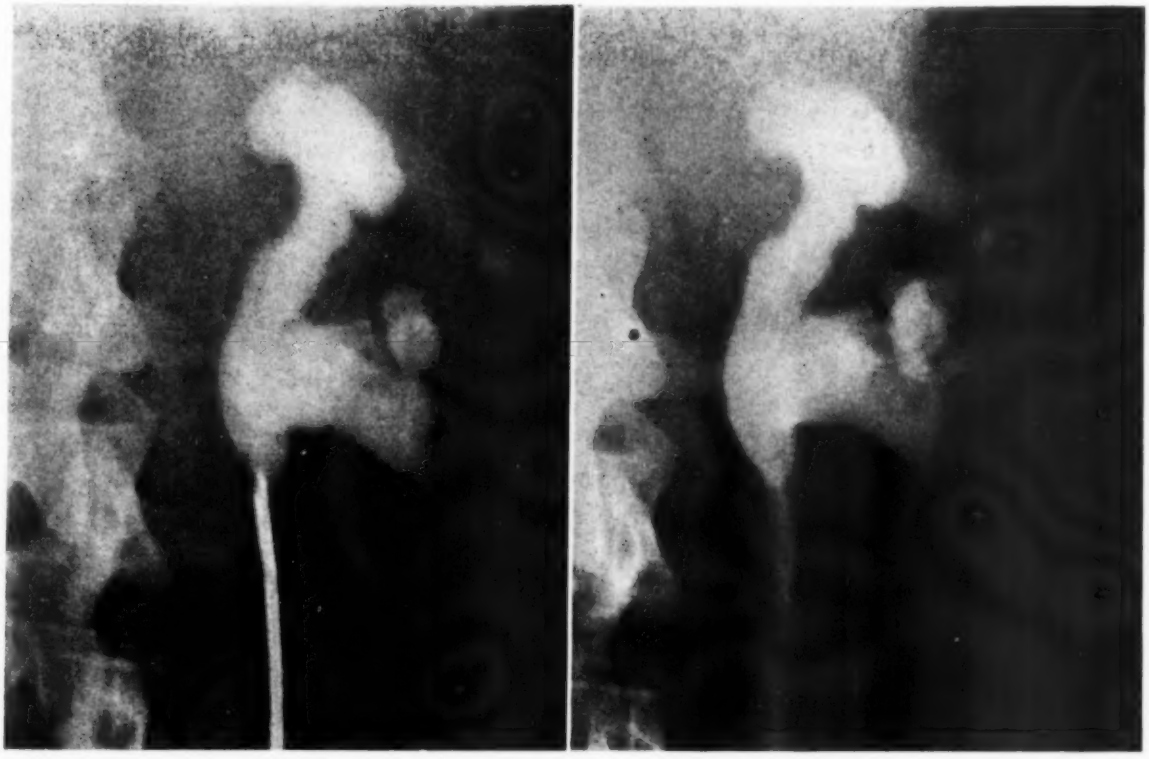
Second Operation. September 10, 1933, a right "V" type nephrolithotomy was done with hemostasis by intestinal clamp on the renal pedicle. The pedicle clamp was loosened every eight minutes. A large staghorn calculus was removed.

Stone Analysis. Left kidney stone, calcium oxylate, calcium magnesium phosphate, triple phosphate. Test for mercury, arsenic, bismuth and iodine negative.

Pre-Operative Urine Report. Right kidney sediment; 50 w.b.c.—5 r.b.c. Right kidney

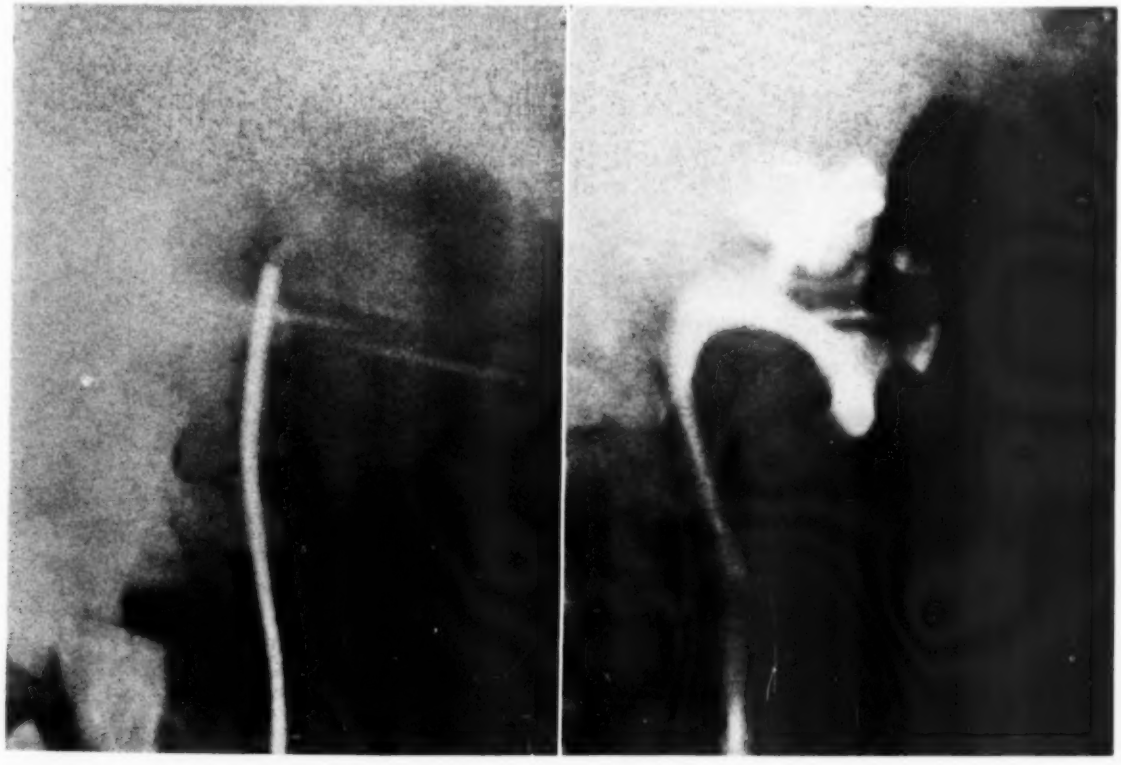
Pre-Operative X-ray Data. Bilateral branching calculi filled the pelvis and calices on both sides. Intravenous pyelography showed the pelves slightly dilated and calices definitely blunted on both sides. Left ureter normal; upper two-thirds of right ureter dilated.

Post-Operative Urine Reports. September 1, 1933. Left kidney sediment; 4 w.b.c. Left kidney function, 5 per cent. January 18, 1934. Left kidney sediment; 100 w.b.c. Left kidney function, 2 per cent. Right kidney sediment; 80 w.b.c. Right kidney function, 7 per cent. December 26, 1936. Left kidney sediment; rare w.b.c., *pH* 5.5. Left appearance time four minutes; left kidney function, 10 per cent. Right kidney sediment; many w.b.c., B. proteus. *pH* 8.0. Right appearance time, four minutes. Right kidney function, 15 per cent.



A B

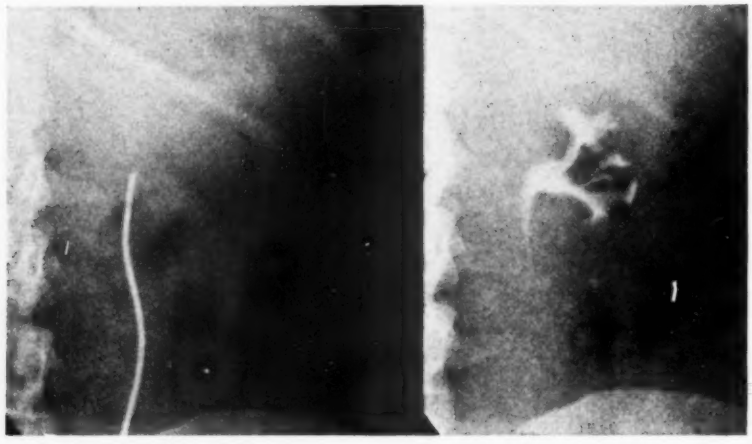
Fig. 14. Case iv. Left kidney. A, pre-operative K. U. B. plate. B, pre-operative retrograde pyelogram.



A B

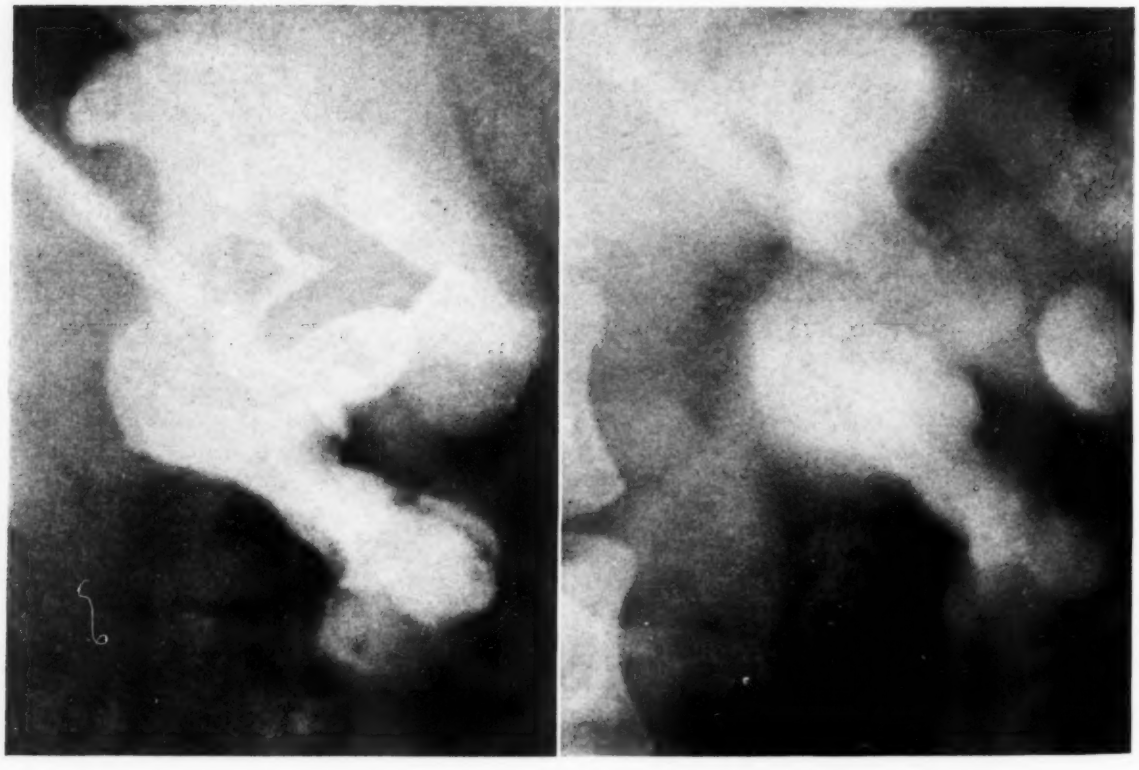
Fig. 15. Case iv, Left kidney. A, K. U. B. plate fifteen months after operation. B, retrograde pyelogram fifteen months after operation.

Post-Operative X-ray Data. September 1, 1933, left pyelogram showed no stone formation in the region of the left kidney. There was an irregular compression of the upper part of the ureter. January 18, 1934, right pyelogram showed a small calcified area in the lower portion of the right kidney. The irregular pelvis was considerably smaller than on previous examination. November 8, 1934, intravenous pyelography showed both kidney outlines visible, rather large. There was a small calculus in the lower pole of the right kidney. The pelves were small, intrarenal in type. The calices were dilated, more especially on the left. Decem-



A B

FIG. 16. Case IV. Left kidney. A, K. U. B. plate three years and two months after operation. B, retrograde pyelogram three years and two months after operation.

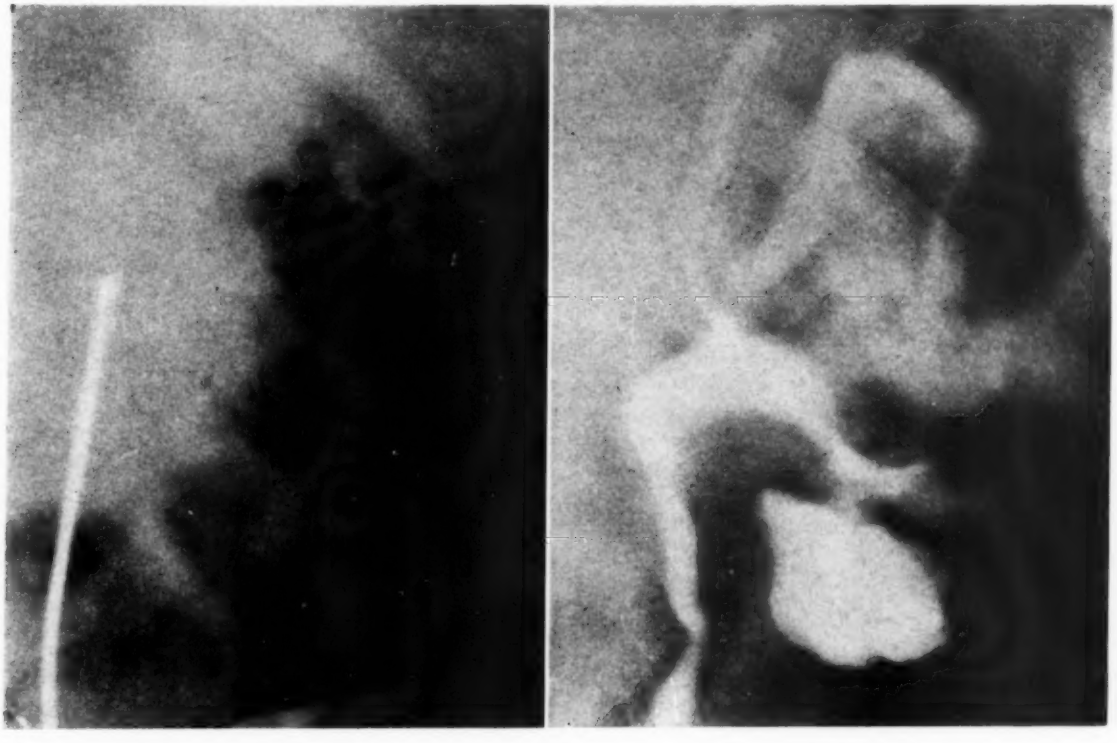


A B

FIG. 17. Case V. Left kidney. A, pre-operative K. U. B. plate. B, pre-operative retrograde pyelogram.

ber 26, 1936, a plain film showed a large, branching calculus filling the right kidney. There was a small area of calcification overlying the middle portion of the left kidney. The right pyelograms showed some dilatation of the upper ureter. The left pyelogram showed dilated calices in the upper pole. The kidney was incompletely filled. (Figs. 8, 9, 10, 11, 12 and 13.)

Post-Operative X-ray Data. July 11, 1933, a left pyelogram showed no evidence of stone and no abnormalities except incomplete filling of the lower minor calyx. May 18, 1935, there was no evidence of calculi in the urinary tract. A left pyelogram showed the pelvis to be intrarenal in type, but normal in size. The calices revealed no definite abnormality. (Figs. 14, 15 and 16.)



A B

Fig. 18. Case VI. A, K. U. B. plate eight weeks after operation. B, retrograde pyelogram eight weeks after operation.

Case 4. H. C., age 30 years.

Operation. March 5, 1932, a left nephrolithotomy was done with intestinal clamp on the renal pedicle for hemostasis. The clamp was loosened every ten minutes. A large, white, branching calculus was removed from the pelvis and upper calyx. Four additional stones were removed.

Pre-Operative Urine Report. Left kidney sediment; 4–5 w.b.c., gram positive staphylococci. Left kidney function, 6 per cent.

Pre-Operative X-ray Data. A large branching calculus filled the entire pelvis and some of the calices of the left kidney. There was blunting of the calices distal to the stone on the pyelogram.

Post-Operative Urine Report. May 18, 1935. Left kidney sediment; negative. Left appearance time, three minutes.

Case 5. R. M., age 27 years.

Operation. July 17, 1933, a left "V" type nephrolithotomy was done with an intestinal clamp on the renal pedicle for hemostasis. The clamp was loosened every eight minutes. Stones were removed, but an *x*-ray in the wound showed one good sized stone remaining in the middle calyx. This stone was removed through a nephrotomy incision on the anterior surface of the kidney.

Stone Analysis. Calcium magnesium phosphate, calcium oxylate. Weight: 65 Gm.

Pre-Operative Urine Report. Left kidney sediment; not reported. Left kidney function, trace.

Pre-Operative X-ray Data. The left kidney was large, and the entire pelvis and calices were filled with a large branching calculus. Intravenous pyelogram disclosed that the right

pelvis was normal in size. There was no excretion of dye through the left kidney.

Post-Operative Urine Report. September 1933, left kidney function, trace. May 28, 1935, left kidney sediment; many bacteria, 6–10 w.b.c. Left kidney, urine culture, B. proteus. Left appearance time six minutes. Left kidney function, 2 per cent. January 4, 1937, left kidney sediment; moderate number of bacilli, 1–3 w.b.c., *pH* 7.5. Left appearance time four and one-half minutes. Left kidney function, 6 per cent.

Post-Operative X-ray Data. September 1933, K.U.B. plate showed a small calcified body in the mid-portion of the left kidney. Pyelograms showed the pelvis to be of the extra-renal type, the calices indefinitely outlined, the opaque media appearing to stand out to the periphery. May 28, 1935, several calcified bodies appeared scattered throughout the left kidney. Left pyelograms showed marked dilatation and irregularity of the calices. The lower calyx was mottled. January 4, 1937, there was an area of calcification over the lower pole of the left kidney. Left pyelograms showed a small renal pelvis with marked dilatation of the calices. (Figs. 17, 18 and 19.)

Fig. 19. Case v. Left kidney. A, K. U. B. plate one year and ten months after operation. B, retrograde pyelogram one year and ten months after operation. C, K. U. B. plate three years and six months after operation. D, retrograde pyelogram three years and six months after operation.

From the follow-up data on the six kidneys operated on approximately three to five years ago, one may regard them as a group and give certain conclusions. All of the five patients are in good general health. None has pain or symptoms referable to the operated side. Four of the five are working. The one who is not at work is the sixty-four year old man who has had his prostate, his gall-bladder, and a bladder stone removed since the nephrotomy for the staghorn renal calculus.

Stone formation has not occurred in the opposite kidney following operation. There has been marked recurrence of stone

formation in two of the six kidneys which were operated on. The present urinary *p*H of one kidney, which has extensive stone recurrence, is 8.0. No determination has been made in the other case. There is no ureteropelvic obstruction in these two cases. In both of these patients, there is renal infection due to B. proteus. The one patient in this group who had the same type of operation on both of his kidneys, within a period of three months, has one kidney free of stone with a urinary *p*H of 5.5, in contrast to a urinary *p*H of 8 from the kidney which shows the recurrent stone.

Four of the six kidneys are known to have been infected before operation. In the four kidneys with little or no recurrence of stone there is no renal infection in two, and a mild bacilluria without pus in the other two. The *p*H values in two of those which have remained free of stone are given as 5.5 and 7.0.

I know of no data concerning the late results following the sagittal section type of nephrotomy for staghorn calculi, and therefore I cannot compare results.

It does seem, however, that the physiologic and anatomic results over a three to five year period, combined with the absence of symptoms, justify the use of this operation for patients with staghorn calculi when it seems advisable to operate and yet necessary to avoid nephrectomy.

None of these patients has been on an adequate medical program for the stone condition since operation. With increased interest and knowledge regarding the theories of stone formation and prevention of recurrence, and with the intelligent application of the newer urinary antiseptics, our results following renal surgery for stone should be definitely improved.

SUMMARY

"V" type nephrotomy, with hemostasis provided by a temporary pedicle clamp, offers an excellent method of removing staghorn calculus in patients where it is necessary to avoid nephrectomy. In this small series there has been definite recurrence of stone in 33 per cent.

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MEDICAL AND SURGICAL TREATMENT OF CHRONIC PROSTATITIS

LOUIS M. ORR, II, M.D.

Chief, Urological Service, Orange General Hospital

ORLANDO, FLORIDA

OF all the pathologic processes in the urinary tract the urologist is called upon to treat, there is perhaps none more resistant to the ordinary measures of therapy than long-standing chronic infections of the prostate gland and its closely associated organs, the seminal vesicles.

By virtue of its structure, both anatomic and histologic, and its location, it becomes, not because of its function but because of its susceptibility to gross changes of great consequence, the most important accessory gland in the reproductive system of the male. The choice of any type of medical therapy should be largely governed by the changes wrought in the normal tissues by the disease process. A clear conception of any pathologic process is dependent upon a familiarity with the normal structure of the organ in question. For this reason it seems relevant to review at this time the developmental anatomy and histology of the prostate gland.

The gland develops from multiple outgrowths of the urethral epithelium, both above and below the openings of the ejaculatory ducts. These outgrowths eventually enclose the prostatic urethra and the ejaculatory ducts as a five lobed, glandular organ, the size of a horse chestnut. The adult prostate gland is firm and elastic and measures about 3.5 cm. in length, 4.5 cm. in width, and 2.5 cm. in thickness. The lobes are encapsulated by a tough fibromuscular membrane which sends prolongations into the interior of the gland, pointing toward the urethral canal, thus dividing the lobes into pyramidal-shaped segments. The interstitial tissue of the prostate is quite pronounced and very dense, comprising more than one-half the total mass of the gland. This interstitial tissue contains a supporting network of connective tissue interlaced with elastic tissue networks and a large number of smooth muscle fibers arranged in layers of various thickness.

Because of the arrangement of its glandular structure, the prostate is classified as a compound tubulo-alveolar organ with two types of secretory glands. These are designated as the external or true prostatic and the internal or periurethral glands. The external glands so greatly predominate as to render the internal ones relatively insignificant. These glands are arranged concentrically about the urethral canal as a center and all point in a posterior curve downward, to empty into the fossa of the prostatic urethra on either side of the verumontanum. Lowsley, in his work on the histology of the prostate published in 1911, estimated that there exist between fifty and seventy of these duct orifices in the normal prostatic urethra. The periurethral, or internal secretory glands of the prostate all lie within the confines of the mucosa and submucosa of the urethral canal. The mucosal glands are comparable in structure to the glands of Littré of the anterior urethra, and are located in the roof of the prostatic urethra immediately over the verumontanum. The submucosal glands are fairly long, branched glands which curve posteriorly and empty into the floor of the prostatic urethra, along with the major excretory ducts of the prostate. A small group of glands which is located just beneath the vesical neck is known as the subcervical group of Albarran. Still another group first described by Home, is found

beneath the trigone. Both of these groups are important, as their enlargement results in the formation of the sixth or subcervical prostatic lobe of Albarran or of the seventh or subtrigonal lobe of Home. Of all these groups of glands, the periurethral, or inner group, is probably of the greatest importance, as investigators such as Tandler and Zuckerkandl, Jacoby and others have long held that hyperplasia of the prostate arises from these glands and not from the larger, more deeply situated, prostatic glands. This would explain why hypertrophy of the prostate is most usually central in origin, that is, around the urethra.

The function of the various groups of glands of the prostate is the production of a thin opalescent liquid with a slightly alkaline reaction and having the peculiar odor of semen. This normal prostatic secretion contains fat droplets, cholesterin, fatty acids and lecithin, which drain into the prostatic urethra through the prostatic ducts. These ducts, although they have no sphincter muscle, are surrounded all along their course to the urethral opening by smooth muscle fibers.

The principal avenue of entrance of infection into the prostate and seminal vesicles is by direct invasion of the prostatic and ejaculatory ducts once the infection has involved the posterior urethra. It continues throughout the glandular structure of the organ, causing the production of leucocytes and lymphocytes which pass through the walls of the acini into the interstitial tissue. The acini and ducts thus become filled with pus and debris, and are unable to empty their contents into the urethra through their single duct openings because of the inflammatory swelling of the contiguous tissues. As resolution of the acute process begins, the reduction in congestion allows better drainage of the acini unless the duct is narrowed or obliterated by the formation of fibrous tissue. The blood stream aids in this resolution by absorbing as much as possible of the products of phagocytosis, but in the vast majority of instances, interference to drainage of the prostatic ducts has taken place and a state of chronic infection develops in the gland.

Other avenues of invasion to be considered are by way of the blood stream from some distant focus of infection or through lymph channels bearing infection from the lower intestinal tract. The onset of the infection by these routes is apt to be insidious in nature. It is most commonly recognized as a *chronic* prostatitis when the patient presents himself for examination, although at times a definite *acute* prostatitis may be the first stage of the infection.

No effort is made here to differentiate the various types of bacteria which invade the lower genital tract. Suffice it to say that by far the greater number of cases of chronic prostatitis have their origin in an acute infection induced by the gonococcus, which, however, is usually replaced later on by other pyogenic bacteria. In over one hundred cultures of the prostatic secretion taken through an endoscope at the University of California by Mathé and others not a single growth of gonococci was obtained. The majority of organisms cultured were of the staphylococcus and streptococcus groups, with the colon group of lesser frequency. The penumococcus has been reported by Culver as being a cause in some cases. McCarthy and Ritter, in 1932, cultured the secretions obtained from the seminal vesicles in thirty patients by direct catheterization of the ejaculatory ducts, and found no evidence of the gonococcus present. It is generally conceded that of all the types of prostatic infection at least 25 per cent to 30 per cent are non-gonococcal in origin; even in those patients with definite gonorrheal histories the gonococcus is supplanted in time by some other type of organism.

The symptoms of chronic prostatitis are usually easily recognized. The individuals who present themselves with a chronic infection of the gland vary in type according to the length of time the infection has been present. After many months or even

years of treatment, they become so changed in mental outlook by the apparent futility of ordinary treatment, that a sexual or genital neurasthenia frequently develops.

An excellent gauge as to whether or not the prostatic ducts are open is provided by a careful study of the prostatic secretion. If the normal elements of the secretion, such as lecithin bodies which are produced in the acini, are lacking in repeated examinations of the secretion, it is quite safe to assume that the prostatic ducts are occluded. A return of lecithin bodies in the secretion indicates improvement in drainage and is of great value in estimating the results of treatment.

Discussion of the therapy of long standing chronic prostatitis is divided under the headings of medical or conservative treatment and surgical or radical treatment. The treatment of the closely allied seminal vesiculitis must also be considered. The routine measures of treatment employed by both the general practitioner and the urologist consist chiefly of more or less frequent prostatic massage associated with the passage of sounds and Kohlman dilators, and followed by the instillation of various silver preparations, chiefly argyrol and silver nitrate. In the average case of prostatitis of fairly recent origin, where the normal structure of the gland has not been altered by the production of excessive connective tissue and drainage is ample, these procedures will usually give satisfactory results. In addition to these office procedures, the patient should exercise great care in personal hygiene, abstain from over-indulgence in sexual intercourse and should be cautioned as regards general constitutional measures that will assist in increasing his body resistance to overcome the infection. The use of heat in all of its various forms plays a very important rôle in assisting in the resolution and absorption of the infectious process in the tissues of the gland. Its field of usefulness as a curative agent is often as important as the use of prostatic massage. In a recent publication by Lewis, the value of the Elliott heat regulator in the treatment of prostatitis has been greatly stressed. I have seen many instances of acute and subacute prostatitis in healthy individuals who, because of the pain produced by massage, were subjected to hydrotherapy alone, in the form of hot sitz baths and rectal irrigations of hot water, and I have seen them make remarkable recoveries. There have been other individuals presenting prostatic infections who, with only a few gentle massages, returned for a check-up six months to a year afterwards with scarcely a leucocyte present in the expressed secretion, and an abundance of lecithin bodies present.

Intravenous medication as an adjunct in the treatment of prostatitis has many enthusiastic advocates. Intravenous injections of 15 c.c. or more of mercurochrome in 1 per cent solution has been reported to give very favorable clinical results. Neoarsphenamine is recommended by some, particularly in the so-called nonspecific cases of prostatitis.

The operation of Belfield, vasostomy, for many years held a prominent place in the treatment of intractable prostatitis and seminal vesiculitis. Solutions of argyrol or mercurochrome injected through the vas deferens into the seminal vesicles oftentimes produce brilliant results in cases of vesiculitis resistant to other modes of therapy. The development by McCarthy of an instrument for catheterizing the ejaculatory ducts has caused the open type of operation to be largely replaced, but in many instances chronic inflammatory changes in the verumontanum have so deformed the ejaculatory duct openings that successful catheterization of the ducts may be very difficult even in the hands of the most expert.

Intraprostatic injection has been much in the forefront in recent years as a procedure of great value, and marked improvement in symptoms has been reported by such men as Townsend, Grant and others. The basis of this method would seem at first quite logical, and one which

might be expected, to give favorable results. The injection of aqueous mercurochrome or other solutions, is made directly through the perineum into the substance of the gland, and the antiseptic action of the drug is thus retained in the gland by the capsule for a considerable length of time.

All of these measures are valuable as to specific action in particular cases of prostatitis, but not all are suitable or effective in every case. Some individuals respond favorably to only one of these measures and others show an indifferent response when all are used. It is these resistant cases of chronic prostatitis, who after many months and often years of various kinds of treatment show no improvement in their condition, who need some type of more radical treatment. Many writers have estimated that this type of prostatitis constitutes only from 5 to 10 per cent of the total number of all prostatic infections, fortunately a relatively small percentage. These cases are, however, of sufficient importance to warrant a search for more effective methods for their relief. It is this group to which the so-called surgical treatment seems so highly adaptable. When correctly applied, surgery yields, in my opinion, the most beneficial results so far obtained in this category.

Thompson, Cook, Collings and many others have reported the use of the electrotome and fulgurating electrode for probing and opening chronic abscessed areas in the posterior urethra, with satisfactory results. These localized areas of swelling and suppuration undoubtedly represent the occlusion of some of the major prostatic drainage ducts. Cutting into these areas allowed better drainage from the deep prostatic glands with gradual amelioration of symptoms and an improved condition of the gland. Thompson further advises the use of the punch to excise segments of the prostatic urethra which has become sclerosed, thus further assisting in opening up the drainage ducts of the gland. He reports a rather large number of patients who were greatly benefited by this procedure alone.

After considering this method of treatment, it appears logical that surgical excision of those areas in the prostatic urethra through which the prostatic ducts drain would aid in eradicating the infection. The mucous membrane of the prostatic urethra in these cases of old prostatitis, when seen through the panendoscope, shows clearly the effects not only of the infection itself, but of the trauma from repeated instrumentation and the injections of chemicals. There is that rough, irregular appearance of the mucosa with occasional follicular swelling and small cysts present. Often some of the ducts are patulous and exude a thick-formed shred of mucus and pus. The membrane is more often of a dusky red color and appears distinctly unhealthy.

In patients of 40 years of age or over, and occasionally in those younger, a definite bulging of the lateral walls of the prostate is sometimes quite noticeable giving the endoscopic appearance of a beginning lateral lobe hypertrophy. Pomeroy, Hirsch and others have called attention to this very condition and have established definite evidence regarding the relationship of infection in these ducts and glands and hypertrophy of the prostate. It is their feeling that true hyperplasia of the gland has its indirect origin from inflammatory reactions which begin at first in the glands nearest the prostatic urethra.

After considering all the factors relating to the chronically infected prostate, I have recently been following a plan of treatment which has proved highly satisfactory and which I consider to be of great assistance in relieving a majority of these rather hopeless cases.

When it has been definitely established by a careful history and a thorough urologic examination that the patient belongs to the group of resistant prostatic infection, a resection of the lateral walls and floor of the prostatic urethra is advised. By using a fine wire loop in the

resectoscope, the flat sections of tissue can be planed away at the discretion of the operator. The use of any type of electrocoagulation should be avoided, as the resulting necrosis and scarring would defeat the primary purpose of the procedure. In younger men only a depth of a few millimeters of tissue need be excised. This incision will cut the prostatic ducts at right angles below the submucosa and thus produce a new duct orifice into the urethra. Usually, two sections of about five-eighth to seven-eighth of an inch in length, taken on either side of the verumontanum and one section in the urethral floor, will open up the majority of the major prostatic ducts. It would probably be better to use some type of cold blade of razor-like sharpness in place of the wire loop to excise the sections, thus eliminating any tissue destruction by the electrical current. Thompson manipulates his punch instrument with great precision and dexterity far better than the majority of us are capable of doing, but visualization through his instrument is inferior to the excellent view afforded by the panendoscope. I am firmly convinced that the ultimate resectoscope will combine the features of both the Thompson and the original McCarthy instruments, with the cutting done entirely with a cold blade.

A few days after the patient has undergone a resection of the sclerotic tissue of the prostatic urethra, he is given daily applications of heat to the prostate. The most satisfactory and the most efficient source of heat I have yet found has been from the Elliott machine. It provides a very accurate control over the degree of heat administered, by virtue of its thermostatic control. These treatments are carried on from forty-five minutes to one hour each day with the temperature ranging from 125 degrees to 130 degrees, but never higher. This local heat is administered daily for fifteen to twenty-five days. The large rectal applicator thoroughly covers the area of the prostate and extends well up over the seminal vesicles. I feel that this application of heat is a factor of tremendous importance in increasing the supply of blood to the prostate and vesicles, thus aiding in the resolution of the inflammatory process. The patients usually complain considerably of weakness and general ill feeling for the first ten or twelve treatments and caution regarding proper rest for at least one hour following treatment must be given. During the second and third weeks of treatment they begin to feel markedly improved, the urine begins to clear of shreds, and gentle prostatic massages at frequent intervals are instituted. Toward the end of the third week very careful Kohlman dilatation is begun, each time followed with a through-and-through irrigation of the urethra. Smears of the prostatic secretion usually begin to show a marked reduction in leucocytes with a return of lecithin bodies, and the prostate becomes more normal in consistency. After the completion of applications of heat through the rectum, the patients undergo a weekly prostatic massage until the leucocyte count of the prostatic secretion approaches normal limits.

Following this plan of therapy, an examination at the end of the first three to six months shows the prostate to be definitely smaller in size. It may be said to have undergone prostatic resorption, as has been reported by many observers following resection of the hypertrophied prostate. The probable explanation of this regression in size of the gland is the release of intraprostatic tension by the process of opening the ducts and allowing better drainage of the prostate.

Although the results so far have not demonstrated a clinical cure in every case, the improvement in the prostate and the amelioration of symptoms has been remarkable, and to my mind this combination of treatment has given most satisfactory results. It may be that along these lines a greater hope of cure may be offered the patient with chronic prostatitis.

THE CLINICAL USE OF ALKYL-DIMETHYL-BENZYL-AMMONIUM CHLORIDE (ZEPHIRAN)

A PRELIMINARY REPORT

CHARLES S. WHITE, M.D., J. LLOYD COLLINS, M.D., AND HOWARD E. NEWMAN, M.D.

WASHINGTON, D. C.

WITHIN the past several years our concept of the bactericidal properties of antiseptic solutions and their use in surgical procedures has undergone a decided change. While the action of some of the newer antiseptics and germicides may not be thoroughly understood, their efficacy and non-toxic action has given them first rank. In this category falls a new type of antiseptic, a mixture of alkyl-dimethyl - benzyl - ammonium chlorides (zephiran).

The product is obtained as a mixture of homologues in which the alkyls represent the radicals derived from the fatty acids of coconut oil. This compound is an amber-colored solid of soap-like consistency. The mixture is quite uniform in composition, and is fully soluble in water, alcohol, and acetone. The aqueous solution is slightly alkaline, possesses a faint aromatic odor, and imparts a soapy sensation to the skin. Germicidal solutions for clinical use have a surface tension of approximately 36 dynes/cm.

Pharmacologic studies involving oral toxicity and effect on the skin and mucous membranes of the laboratory animals revealed that this compound in germicidal concentration is relatively non-toxic and non-irritating.[1]

Heineman[2] has contributed a concise but thorough evaluation of zephiran. His studies embraced the following: (1) phenol coefficient determination; (2) inhibition and penetration tests; (3) tests for disinfection of the human skin; and (4) destruction of spores of bacteria and fungi.

The phenol coefficient was determined by the method as described by the Food and Drug Administration. Against Staphylococcus aureus the phenol coefficient was 275, against Eberthella typhosa 176, and against E. coli 318. Somewhat higher figures under the same conditions were obtained by Dunn[3,4] and Deskowitz.[1]

In penetration and inhibition tests, using the agar cup-plate method, the results were convincing and paralleled the phenol coefficient tests. The largest zones were formed by the Staphylococcus aureus, the smallest by the Streptococcus pyogenes.

Ten per cent alcoholic and 10 per cent aqueous zephiran solutions were used in skin disinfection tests, which were carried out in the following manner: After the hands were scrubbed with a brush, soap and water for eight minutes, they were dried with a sterile towel and the fingers immersed five minutes in the test solution and then washed with sterile water. Agar was liquefied, poured over the fingers, and collected in sterile petri dishes. No colonies could be detected in five days. This confirms the work of Domagk,[5] which was carried out under similar conditions.

Tests were made with the object of determining the destructive effect of a solution of the ammonium chloride compound on spores of Bacillus subtilis, Trichophyton gypseum, and a Mucor, which was isolated from milk. The results showed that in a concentration of 1:500 (distilled water) the ammonium chloride compound destroyed the spores of Bacillus subtilis and those of the two types of fungi in five minutes.

With the facts of an extensive bacteriologic background before us, we felt that a clinical test was warranted. It has been truthfully stated that no amount of laboratory tests can replace actual clinical experience in evaluating a germicide. There is no test-tube comparable to living tissue for

this purpose. There is no test that can replace the routine use of a solution over a considerable period of time, embracing as it must, many variable conditions, unapproached by any laboratory.

We inaugurated a skin test which, in spite of its inherent faults, we considered as the nearest approach to an actual clinical test. The surgeons, assistants and nurses scrubbed up in the usual manner, except that the ammonium chloride compound solution 1:1000 (Tincture zephiran) was used for immersing the hands and forearms, instead of the usual mercurial solution and alcohol. In every case, immediately before operation, a small piece of full thickness untreated skin was removed with sterile forceps and scalpel and placed in a sterile test-tube marked in code in order that the bacteriologist could not possibly know anything of the treatment of this specimen. The surface to be operated upon was then scrubbed with soap and water, benzine, and alcohol. When the surface was dry, tincture of zephiran 1:1000 was well applied with a sterile sponge and allowed to dry. Then a small piece of full thickness of skin was removed, dropped into a sterile test tube, marked in code, and sent to the laboratory.

We shall designate these sections of skin as "control" and "treated." There were fifty-one such cases and the skin, in most instances, was removed from the abdomen. In the fifty-one cases in which cultures were made from the untreated skin by excising a piece, there was growth in every instance, as follows:

Staphylococcus albus	35
Staphylococcus albus and aureus	5
Staphylococcus albus and citreus	2
Staphylococcus aureus	3
Staphylococcus albus and Streptococci	4
Budding yeast	1
Gram-positive cocci in chains	1
	51

In the area treated by zephiran, in which a piece of skin was removed, there was growth in two cases: (1) Gram-negative bacillus: a spore-bearing bacillus and spores; and (2) Gram-positive coccus in chains and clusters. The organisms appeared considerably smaller than those in the control.

Of the fifty-one cases, forty-six were of the abdominal type, four were breast tumors, and one a lipoma of the chest wall. They were without exception "clean cases," that is, no operation was done in the presence of known skin infections. There was but one infected wound in this series: this case was a cholecystogastrostomy for carcinoma of the head of the pancreas. The wound was definitely infected, but in all probability the source of contamination was from the field of operation rather than from the skin. There were twenty-one additional cases in which zephiran was employed in skin preparation, but for very good reasons (many of these patients were employees of the institution) no skin was removed for laboratory tests. These cases all healed by primary union. In all, there were seventy-six cases of zephiran preparation with one wound infection. No irritation was noted on the skin of the patient or on the hands of the operator, assistants, or nurses. No pustulation or other evidence of irritation was noted on the skin on which zephiran was used and upon which adhesive plaster was applied.

Our percentage of infections in this hospital, and in fact in most of our work elsewhere, is very much higher. This has occurred in spite of the fact that we require skin preparation for operation on two successive days unless the operation is an urgent one and time will not permit delay. The incidence of one infected wound in seventy-six cases, while representing a small series, is very gratifying to us. It is all the more remarkable when the type of patient is considered. They were all indigents, on the ward of a municipal hospital, many feeble, poorly nourished, a fair percentage of them emergency cases, and many had not availed themselves for unknown periods of the usual sanitation afforded by soap and water.

To check our results, we requested the gynecologic division to carry out a series of cases using the same details in skin preparation with 3½ per cent iodine as the germicide. There were twenty-five such cases, all abdominal operations. In the control biopsies there was growth in every case. No biopsies were taken from the iodine-treated skin, but seven infected wounds were present in the twenty-five cases.

CONCLUSIONS

The reduction in wound infection, judging by our experience over a long period with many other antiseptics, is too striking to be termed an accident or a fortuitous circumstance. While the number of cases in which zephiran was used was a comparatively small one, there are sufficient data to warrant the statement that it is a safe and effective germicide and justifies an extended trial.

SUMMARY

In a series of seventy-six cases, using zephiran as a skin germicide, wound infection followed in one case, an incidence of 1.3 per cent. There is reasonable doubt about skin contamination in this case.

In fifty-one cases from skin cut from areas prepared with zephiran, cultures showed growth in two cases. Growth was present in all controls.

In a comparative study of twenty-five cases in which laparotomy was performed with 3½ per cent tincture of iodine, there were infected wounds in seven cases or 28 per cent.

No skin irritation was noted with zephiran even under adhesive plaster.

We wish to express our thanks to Dr. H. H. Leffler for his valuable laboratory work.

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EVISCERATION—A POST-OPERATIVE COMPLICATION

ISIDOR KROSS, M.D., F.A.C.S.

Attending Surgeon, City Hospital; Associate Attending Surgeon, Beth Israel and Montefiore Hospitals

NEW YORK CITY

AN investigation was made of the post-operative eviscerations that occurred in the surgical and gynecological services of the Beth Israel Hospital from 1930 to 1936. By evisceration is meant, in this report, not only wound rupture, but also an accompanying protrusion of omentum or intestine. During this period 4,433 abdominal operations (not including inguinal or femoral hernias, or suprapubic prostatectomies) were performed, and evisceration occurred in forty-four of these cases. The frequency of occurrence was just a fraction more than 1 per cent. Sex apparently played no rôle as an etiologic factor, since twenty-four of these occurred in the female patients, and twenty in the male. Since the gynecological patients were included in this series, the slight preponderance among the females is thus readily accounted for.

Numerous investigations have been made and reports on the frequency of this complication have been published, but there is still lacking a satisfactory explanation for its causation and development. The theories generally advanced are: (1) failure of the suture material; (2) unusual strains; (3) constitutional factors, i.e., asthenia and debility causing a failure of the healing process; and (4) wound infection.

In this series, the age of the patient did not seem to play any rôle, as the major part of the hospital population was between the third and fifth decades. The character of the incision itself has no important part in this condition, and it is well known that the frequency of wound rupture is about the same in most clinics, in spite of the fact that the character of the incision in these different clinics varies considerably. It is also interesting to note that the median suprapubic incision which is used for pelvic operations, showed rupture of the wound in eleven instances where fibroids were removed and in no instance where the more frequent adnexal operation was performed. This may be due to the debility and asthenia brought on by the bleeding which is so common a factor in fibroids.

TABLE I

Age	No. of Cases
20–29	2
30–39	7
40–49	15
50–59	12
60–69	4
70 and over	3

TABLE II

Day of Rupture	No. of Cases
3	1
4	5
5	6
6	9
7	7
8	8
9	4
10	3
11	1
12	1

Before discussing the various theories of the etiology of evisceration, it would be helpful to review briefly the manner of wound healing. This will help to explain the frequency with which this accident occurs between the fourth and the eighth post-operative days. Wound healing was studied in detail by Meleney and Howes.[1,2] These investigators divide the process into two stages, the first or exudative stage lasting four or five days, and the second or reparative stage beginning at the end of the first stage and continuing until the wound is fully healed. There is no sharp, sudden demarcation between the two processes; the second stage begins before the first is fully completed. In the first period an exudate develops. The character and extent of this phenomenon depends upon several factors: the number of dead

and injured cells, the amount of extravasated blood and lymph, the presence of dust and bacteria in the wound, and the amount of suture material employed. During this period, the tensile strength of the sutures diminishes rapidly and the holding power of the tissues becomes less. During the second period, the strength of the wound increases rapidly as a result of the development of fibroplasia with ultimate scar formation. At this time the exudate undergoes absorption. The duration of the first period depends upon the character and extent of the exudate. Tissue necrosis and inflammation not only increase the duration of this period, but also delay the beginning of the reparative or second stage, thus causing tissue strength to be gained more slowly. Since all sutures act as foreign bodies, the quantity of suture material and its bulk are important factors in the healing process. The tightness with which the tissues are coapted, and the tightness with which sutures and ligatures are tied, have a direct bearing on the amount of tissue necrosis that takes place. This factor, which is so frequently overlooked, plays a considerable rôle in wound healing.

In a series of experiments on stomach wounds in rats, Howes[2] showed definitely that all wounds, after the first day, steadily lost strength up to the fourth day. From then on the wounds began to increase in strength, and after about the seventh day the tensile strength of the wound was such that when force was applied by distending the stomach, rupture occurred in the tissues around the wound and not in the wound itself. Up to this time, the same force caused rupture to take place at the site of the wound proper. Howes also showed that with the use of silk sutures, the first stage was of shorter duration than with catgut, and also that the reparative stage began earlier, proceeded more rapidly and reached its greatest strength sooner. He also demonstrated that the exudate lasted longer and that the development of the collagen fibers began later in the wounds where catgut was employed.

As evidence in favor of the theory of failure of the suture material itself, numerous examples have been published in which at secondary operation no trace of the suture material could be found. There is no doubt that this explanation may account for some cases where there is a combination of several factors, such as premature absorption of catgut or tearing of catgut, in the first few days after operation before tissue healing has advanced perceptibly, accompanied by sudden increase in intra-abdominal pressure, especially in the presence of infection. Inspection of Table II, however, shows that many cases occur when tissue healing is quite advanced, where there is no evidence of premature absorption of catgut, and where there is no evidence of infection. In one case, evisceration occurred although the suture material consisted of unabsorbable silkworm gut.

Colp,[3] who reported the results of a thorough investigation of these cases at the Mount Sinai Hospital, holds that the primary disease is undoubtedly the most important factor in the production of evisceration. It is also interesting to note that Sokolow,[4] who also holds this view, called attention to the frequency with which wound rupture occurred among the northern people, especially in the early part of the year. This he ascribed to the lack of vitamin C and to the general fatigue at the end of the very long winters in northern countries.

From the nature of the diseases for which operation was instituted, it seems to be apparant that debilitating conditions constitute a frequent and common association with wound rupture. (Table III.)

In addition to the general asthenia and its interference with normal wound healing, Grace[7] considers sudden increase in intra-abdominal pressure to be the direct and most frequent cause of wound rupture. That there is this relationship can be seen both from the tables he shows in his publication and also from the post-operative factors tending to sudden increase in the intra-abdominal pressure, as shown

in the study of this series of cases. (Table IV.) It is not uncommon for the patient to report that he felt something give way immediately after a spell of coughing or sneezing.

TABLE III

Nature of Disease	No.
Carcinoma	
Stomach	5
Colon	4
Pancreas	1
Liver	1
Rectum	1
Gall-bladder	1
Uterine fibroids	11
Biliary system	8
Peptic ulcer	5
Acute appendicitis	2
Intestinal adhesions	2
Intestinal obstruction	1
Tuberculosis of cecum	1
Hydatid mole	1

TABLE IV

Post-operative Complications	No.
Vomiting	26
Restlessness	20
Distention	19
Cough	16
Belching	12
Nausea	11
Retching	7

In view of the usual suddenness and unexpectedness of the evisceration, it is worth while emphasizing a clinical observation which in many instances gave warning of an impending and as yet unrecognized evisceration. This was the presence of a dressing saturated with bloody serum. This occurred in twenty-four cases of the present series. (Table v.)

TABLE V

Day of Appearance Post-Operative	No. of Cases
4	2
5	2
6	7
7	5
8	5
10	2
11	1

In considering the theory that general constitutional factors are responsible for this condition, two questions immediately come to the fore. How can one explain the fact that the rupture so frequently occurs in one part of the wound only, when the rest of the incision has healed firmly and fully? Again, how, on the basis of this theory, can one explain the firm and satisfactory healing of the same wound when it has been resutured after the rupture has taken place?

Some light is shed on this subject by the recent work of Freeman[5] and King,[6] who showed in a series of experiments that the responsible factor consists of a local condition. Freeman reports the results of his experiments in which a piece of omentum was allowed to protrude through an opening in the peritoneum between the muscles and under the fascia. In a number of cases he found failure of union of the muscle and fascia. His explanation appears rational and logical: the protruding omentum, as a result of pressure, becomes congested and hence increases in size. It then acts as a wedge, and any increase in intra-abdominal pressure (especially in the presence of infection and during the time when the wound healing is still incomplete) will push the piece of omentum through the incision and force it open. King, in a study of post-operative hernis, (which may be looked upon as an incomplete rupture of a wound), comes to a similar conclusion, namely, that the underlying factor is an opening in the peritoneum into which the abdominal viscera find a passage. He concludes that evisceration is a result of increased intra-abdominal pressure plus an incompletely healed wound.

To complicate matters still more as far as this theory is concerned, there arises another question. If the condition is as King and Freeman maintain—a local one due to imperfect closure or healing of the peritoneum—why does it not occur more frequently in those cases where wounds are drained intraperitoneally, especially where they are deliberately left open and packed, as is so frequently done in cases of far advanced appendiceal infections. However, in a series of experiments on rabbits, the author has deliberately left a small opening in the peritoneum, otherwise closing the abdominal wound firmly and fully. While these experiments are still

under way and not yet completed, it has been observed in a sufficient number of instances that, at the site of the deliberately made opening, an evisceration took place and the intestine was found protruding through the opening, while the rest of the wound was fully healed.

In considering all these factors, one must conclude that no single condition by itself is solely responsible for all cases of evisceration. This complication is brought on by a combination of events, in which any single one may by itself be insufficient, but when combined with others may produce wound rupture. The results of the experiments of Freeman, King, and the author, and the frequent findings of a partial wound rupture with healing of the rest of the wound, justify the assumption that in all probability the one most important factor is a defect in the closure of the peritoneum.

As far as prevention of wound rupture is concerned, all the theories discussed have a practical value, which can be summarized as follows:

1. The general condition of the patient should be brought up to the highest point possible, so that the constitutional factors involved in wound healing should be at an optimum level.

2. Asepsis should be maintained most strictly in order to reduce the exudative period to a minimum, and to allow the reparative stage to be begun as early as possible.

3. Special care should be given to the patient to avoid all unnecessary increase in intra-abdominal pressure due to gastric or intestinal distention, by early use of the Levin tube, rectal tube and enemata; to avoid or reduce cough by administration of pulmonary sedatives; and to avoid restlessness by administration of cerebrospinal sedatives.

Last and probably most important, meticulous care should be given to an exact and thorough closure of the wound, with special regard to the use of only an essential minimum of suture material. Care in applying both sutures and ligatures is necessary to avoid strangulation of tissues.

I wish to thank Dr. H. E. Isaacs, Dr. I. C. Rubin, and Dr. M. Robinson for their kindness in granting me the privilege of studying the cases from their services.

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A PLASTIC OPERATION OF THE BREAST WITH FLEXIBLE ADAPTATION*

PAUL W. VESTAL, M.D., F.A.C.S.

Instructor in Surgery, Yale University School of Medicine; Assistant Attending Surgeon, New Haven Hospital

NEW HAVEN, CONNECTICUT

IT is not the purpose of this paper to discuss the literature on plastic operations upon the breast, but rather to report a procedure which, in our hands, has served to bridge a gap left by other methods. For those interested in the subject a complete résumé of the literature may be found in a most able paper by Maliniac.[1]

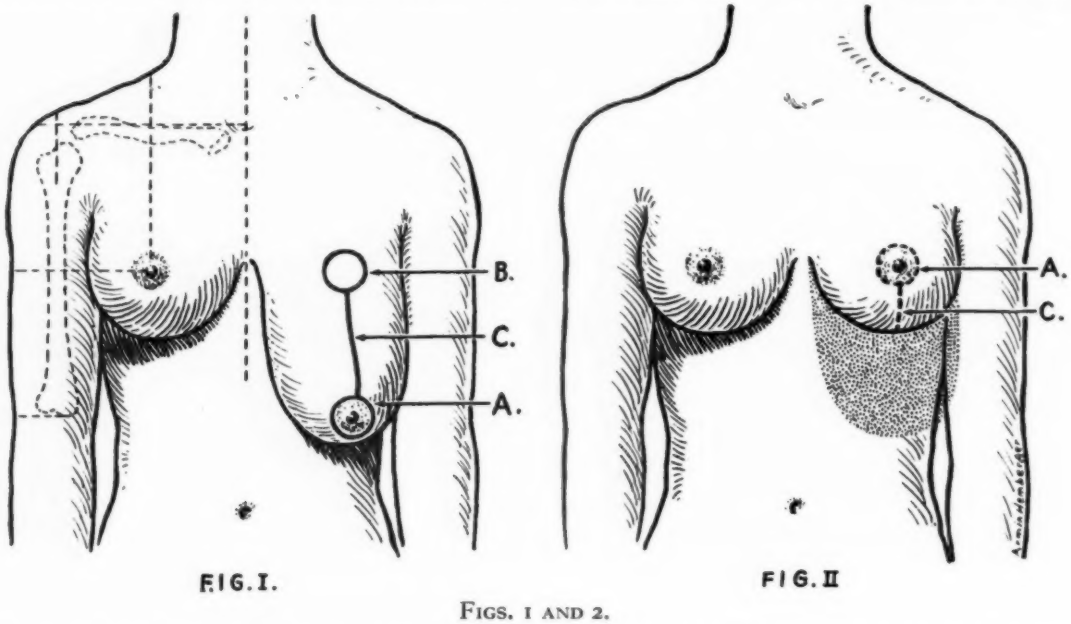

FIG. I. FIG. II

FIGS. 1 AND 2.

Indications for Operation. 1. Benign tumors wherein a considerable portion of the breast tissue must be removed with excision of the tumor.

2. Asymmetry of breasts of a conspicuous degree.

3. Ptosis of the breasts. Usually found in young women, both single and married, due to developmental phenomena or to the result of tight fitting brassières, following in the wake of past styles.

Ptosis of breasts in older women is common and is usually seen in multiparae. Unless tumors are present, operation is not indicated. In younger women, the pathology and subsequent demands for correction are more imperative. Where the breasts are heavy and produce dragging from the shoulders and pain in the anterior chest wall with resultant poor posture, interference is indicated. Furthermore, where abnormal hypertrophy and asymmetry exists, the mental reaction is most pronounced. The conspicuous location of the abnormality makes these individuals self-conscious, retiring, bars them from certain athletic and professional endeavors and goes far towards producing mental disorders. Women are often heard to say that they would prefer to have no breasts at all than be encumbered with the dragging masses of tissue which, unsupported, rest on the abdomen instead of the chest. Their attitude is understandable when one observes the resulting postural defects and mental reactions.

Operation. Maliniac has developed the most adaptable operation for these cases,

* Read before the New Haven Medical Association, June 19, 1935.

based on physiologic and anatomic lines. However, certain occasions arise where his operation is not feasible. In excising tumors and correcting the lesser breast deformities, sufficient skin is not always present in the anterior flap for the carrying out of his procedure without encroaching upon the areola or leaving a conspicuous scar. Consequently, the operation described below was developed.

Fig. 3.

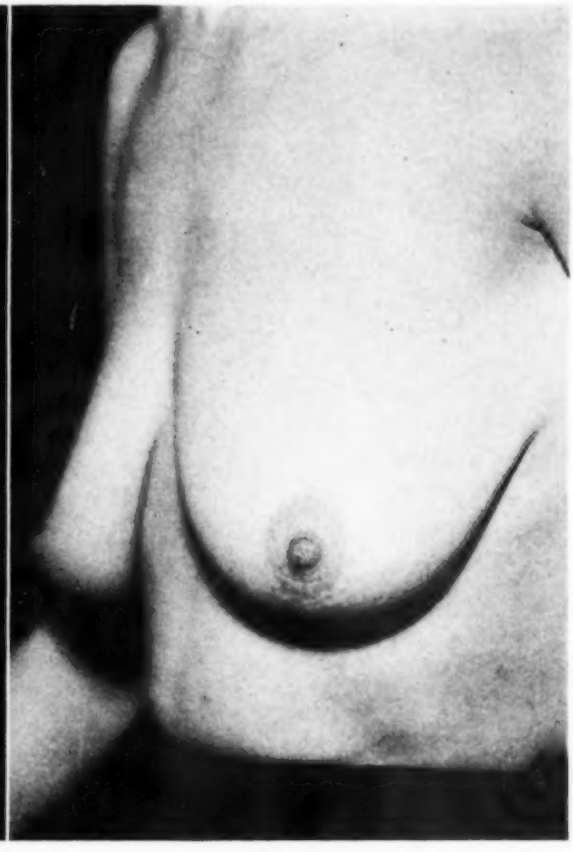

Fig. 4.

Figs. 3 and 4. A member of the younger generation whose breasts were flattened and relaxed by styles of recent years. A moderate cystic mastitis was also present. The breasts were returned to their normal anatomic position and the cysts removed.

1. A circular incision (Fig. 1A) is made around the areola in conformity with the size of the opposite areola (if only one side is to be done). If the procedure is bilateral, the areola may be reduced to any desired size.

2. The future location of the nipple is determined at a point where a vertical line through the midclavicular area intersects a horizontal line from a point at the middle of the humerus. A circular incision of the same diameter as the areola to be transplanted is made at this point (Fig. 1B) in the anterior breast surface. The two circular incisions are then connected (Fig. 1C) by a vertical incision. Suitable undercutting of the relaxed skin is carried out on either side of the vertical incision so that the two side flaps of skin hang free from the breast substance. It is important not to undercut too far and leave dead space. The blood supply to the breast must also always be safeguarded.

3. Where tumors are present, they are excised, and if hypertrophy exists, the excess tissue is excised from the base of the breast, thus protecting the blood supply and galactiferous ducts.

4. The base of the breast is affixed by interrupted sutures to the pectoral fascia, thus giving good support without tension on the skin. This goes far towards preventing a recurrence of the ptosis.

5. The breast with its nipple and areola intact is now located in its proper anatomic position and the areola is sutured to the margins of the skin in the circular opening previously made in the anterior flap. (Fig. 2A.) The long flaps of redundant skin are then cut away in such a manner as to bring their cut margins together beneath the transplanted breast with the single suture line passing downward from the areola to the posterior mammary sulcus. (Fig. 2C.) This gives further support to the breast by a hammock-like suspension. It is usually necessary to cut triangular flaps in the posterior mammary sulcus in order to reduce remaining redundant skin and provide normal shape to the breast. These incisions are covered by the breasts and are not conspicuous. The suture line around the areola is not detectable after a few months. Subcuticular sutures give excellent approximation and reduce the amount of scar on the skin. Rubber dam drains for forty-eight hours prevent hematomata.

FIG. 5.

FIG. 6.

FIGS. 5 AND 6. Pre-operative deformities and post-operative results. The poor posture is incident to the heavy breasts which exert a downward drag on the shoulders. Extensive cystic mastitis was found at operation. Note the transverse sulcus in the abdominal wall prior to operation.

The application of an ice cap immediately following operation aids in preventing effusion and infection.

SUMMARY

A new operation for the reconstruction of the breast along anatomic lines and position, is described. It is indicated after the removal of large benign tumors, in the correction of abnormally pendulous or hypertrophied breasts, and in the adjustment of asymmetry of the mammary glands without disturbance of physiologic function.

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LYMPHATIC INJECTION WITH RADIOPAQUE SUBSTANCE FOR ROENTGEN EXAMINATION IN CARCINOMA OF THE MAMMARY GLAND

A PRELIMINARY NOTE*

JOHN J. GILBRIDE, M.A., M.D.

PHILADELPHIA, PENNSYLVANIA

THE object of lymphatic injection in carcinoma of the mammary gland is to determine, if possible, and so far as possible, a method which would enable the surgeon to arrive at a fairly definite conclusion of the extent of the local malignant disease. In some cases of malignancy of the mammary gland, the disease is limited locally to a distance of 5 cm. or 7 cm. beyond the periphery of the tumor mass. This statement does not apply to the presence of metastasis. Other cases of mammary carcinoma extend along the skin for variable distances, so that it is impossible to ascertain the extent of skin invasion by the malignant process except as the surgeon can apply his experience and judgment. Some of these patients, even after wide excision and radical operation on the mammary gland, show a continuation of the malignant disease in the skin. This is not reccurrence, but a continued course of the malignancy, and no one is to blame for the extension.

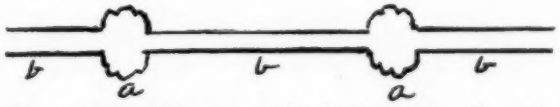

FIG. 1. A semidiagrammatic sketch of a section of a lymphatic vessel (b), and its bulbous expansions (a). This drawing was made after an examination of the lymphatics in the ear of a living rabbit, which was held firmly in position with its ear on the stage of a dissecting microscope.

Efforts have been made to prevent transplantation of malignant cells during the performance of surgical operations; the regular surgical scalpel is sometimes replaced by an electrosurgical knife. The electrosurgical knife has its advantages over the plain surgical knife for the reason before mentioned, but the most important point is that the skin in the region of the incision has been invaded before the operation. Advance in surgical procedure cannot be made until there is available something definite as to the extent and local limitation of the malignant disease.

FIG. 2. Carcinoma of the right mammary gland. The growth was adherent to the skin and an area of retraction of the skin can be seen in the lower inner quadrant of the gland. A silver coin was placed over the largest of three lymphatic nodes posterior to the outer border of the pectoralis major muscle.

The normal lymphatics (Fig. 1) in the ear of a living rabbit were studied by holding the rabbit firmly in a position with its ear resting on the stage of a dissecting microscope. The normal lymphatic vessels, including their bulbous enlargements, change in size and shape in accordance with their state of activity or rest. All the

* Read before the South Branch of the Philadelphia County Medical Society, Dec. 22, 1936.

lymphatics are not active at the same time.

Case Report. A female, colored, aged 64, had a tumor in the right mammary gland for fifteen months. Pain of a darting character, intermittent in type, had been present for the preceding ten months. There was also a burning feeling in the region of the growth. On inspection, the skin was adherent to the underlying mass, and an area of retraction was present over the lower inner quadrant of the mammary gland on the right side. The mass was irregular in outline, 9 cm. in the transverse dimension and 7 cm. in the vertical. It extended from a point 1 cm. to the right border of the sternum, was hard, nodular, tender on palpation, and only slightly movable on the underlying chest wall. There had been no discharge from the right nipple. One large lymph node and two smaller nodes were palpable beneath the right free border of the pectoralis major muscle. A coin was placed over the largest node which measured 3 cm. in the vertical direction and 2.5 cm. in the transverse dimension. (Fig. 2.) These nodes were very hard, irregular in outline, and adherent to the surrounding structures.

Roentgen examination confirmed the clinical diagnosis in that the mass involved almost the entire right mammary gland. It was inoperable.

Fig. 3. Roentgenogram made after injection of radiopaque substance into the lower part of the carcinoma in the right mammary gland. The object on the right is the hand and ring of the attendant holding the breast.

Fig. 4. Roentgenogram showing the radiopaque substance within the lymphatics. Arrows on the right point to the tumor, while those vertically arranged show the lymphatics along the chest wall. Note the similarity in appearance of the lymphatics to the diagram in Figure 1.

Roentgen examination of the left mammary gland, the thorax, vertebrae, and the bones did not show any recognizable signs of metastasis. Physical examination showed the right axillary lymph nodes were invaded.

A radiopaque substance was injected into the lower border of the mass in the right mammary gland as shown in Figure 3. A Roentgen examination followed that procedure and the presence of the radiopaque substance in the lymphatics is shown in the roentgenogram (Fig. 4) which was made on October 16, 1936. A second roentgenologic examination was made on December 21, 1936, and a third (Fig. 5) on December 28, 1936. The radiopaque substance which was deposited into the tissues (Fig. 3) had been taken up by the lymphatics and distributed throughout these structures. (Fig. 4.) The result of examination shows two large lymphatic vessels that can be seen distinctly in the upper part of this roentgenogram. These

vessels vary in width and differ in size from each other, and present areas of dilatation, and others of contraction, a condition which may be seen in normal lymphatic vessels. In some parts the vessels appear to be wide open. They pass upward to the infraclavicular and the supraclavicular lymph nodes. The lymph vessels in the outer part of the gland and those that pass to the axilla were obstructed. There was an absence of the bulbous enlargements in the two superior lymph vessels above mentioned. The lymph vessels that pass downward and inward to the extraperitoneal tissue of the epigastric triangle, showed signs of obstruction. (Fig. 5.) The obstruction in these vessels was not complete because the bulbous enlargements had changed their shape, were somewhat smaller, and contained less of the radiopaque substance. The last roentgenogram was made seventy-three days after the first examination. Another roentgenogram made July 24, 1937 shows the radiopaque substance completely disappeared.

There has not been heretofore, so far as I know, a successful method for injection of a satisfactory radiopaque substance into the lymphatics of a living human being. The substance used here, lympholan, is non-toxic, and its injection has caused no irritation or other harmful condition. The lymphatic vessels have not been damaged by rupture and extravasation of the injected material into the tissues. Satisfactory roentgenograms have been made following its use.

The technique consists of an injection of 0.25 c.c. of lympholan into the tissues in the vicinity of the periphery of the tumor. Two or more injections may be made. For example, an injection may be made in the lower inner zone, and another injection made into the lower outer zone if necessary to cover the areas of lymph drainage. A Roentgen examination is made after the first injection if only one injection is made, or the examination may be made following the second, or the last injection. A second Roentgen examination is made twenty-four hours later, and a third may be made several days after the last injection.

SUMMARY

There are two important questions in the management of patients with malignant disease of the mammary gland: (1) the determination of the presence of metastasis; and (2) the local extent of the disease.

Fig. 5. Roentgenogram of same case as in Figures 2, 3 and 4. Taken seventy-three days after injection of the lymphatics. Radiopaque substance is still present, owing to lymphatic obstruction.

At the present time, answers can be given that are only approximately correct. Injection of the lymphatics of this organ was undertaken to provide a scientific method which would furnish further information of the extent of the local malignant process. Two problems appear to have been surmounted, in that the lymphatics can be injected with a radiopaque substance which can be seen on an *x*-ray film, and that the substance employed does not produce harmful effects. Therefore, scientific methods for the determination of the local extent and limitations of the malignant process offer the best prospects for improved results.

CASE REPORTS

PRIMARY CARCINOMA OF THE FALLOPIAN TUBE WITH REPORT OF A CASE

LOUIS E. PHANEUF, M.D., SC.D., F.A.C.S.

Beth Israel Hospital

BOSTON, MASSACHUSETTS

BARROWS,[1] in 1927, published a comprehensive paper with an extensive review of the literature on primary carcinoma of the Fallopian tube. Since then publications from Smith,[2] Kahn and Norris,[3] Dannreuther,[4] and Robinson[5] have appeared. According to Robinson,[5] primary carcinoma of the oviduct was first recorded by Raynaud in 1847 and the first pathologic description was given by Rokitansky in 1861. Nürnberger should be assigned priority to Orthman, who described this condition in 1886. Since Nürnberger published his collection of 301 cases from 1886 to 1931, Robinson has collected 48 cases from 1931 to 1935, including those omitted by Nürnberger and his own additional two cases. In all there are 349 cases of primary tubal cancer recorded in the literature up to 1935. From the standpoint of the frequency of the lesion, Vest, in 1914, found four out of about 19,000 gynecologic admissions to the Johns Hopkins Hospital, while Barrows reported three cases seen in 30,000 gynecologic patients admitted to the Bellevue Hospital from 1911 to 1927. Robinson states that the frequency of this disorder ranges from 0.03 to 0.31 per cent of gynecologic affections.

The origin of primary tubal carcinoma, according to Doran and Fearn, represents a malignant change occurring in a benign papillomatous condition of the tube lining resulting from a preexisting salpingitis, while Sänger and Barth believe that it appears a long time subsequent to a chronic, probably purulent inflammation of the tubes, usually occurring about the time of the climacteric. O. Frankl, quoted by Robinson, states: "If inflammation would be a precursor of primary carcinoma of the Fallopian tube, then the incidence of malignancy ought to be much higher, for salpingitis is overwhelmingly frequent." Previous inflammation as an etiologic factor, therefore, is disputed.

The age of the patients varies considerably. Thus two women were found to be 18 and two others 70, while the large majority were found in the years between 40 and 60.

Three types of carcinoma of the Fallopian tube have been described in the literature: the papillary, the adenomatous and the alveolar. Some pathologists believe, however, that these are merely morphologic phases in the development of the disease, the papillary being the earliest, the alveolar the latest, and the adenomatous the intermediate stage. Primary carcinoma frequently affects the tubes bilaterally. In view of this fact, Robinson feels that the apparently normal appearance of the opposite tube should not deter from a radical procedure. Metastasis from this malignant disorder usually takes place through the lymphatics. Clinical and pathologic experience have demonstrated that metastasis may be found in distant locations before the tubal wall has become involved; this would tend to be against continuity and contiguity as important factors in the spread of the disease.

The diagnosis before operation is extremely difficult, and the laparotomy is usually performed under a different diagnosis. The main symptoms are pain across the pelvis which may be accentuated on one side or the other, and a vaginal discharge which may be yellow, brown, watery or bloody. The pain frequently takes the form of intermittent cramps which may disappear after the free flow of the discharge; the distended tube empties its contents in the uterine cavity and the vagina and thus relieves its own internal tension. The usual findings on bimanual examination are those of tubal inflammation from other causes and offer little help in arriving at a diagnosis.

The treatment should consist of the ablation of the entire pelvic structures, that is, panhysterectomy with bilateral salpingo-oöphorectomy, as well as the removal of palpable glands. Deep *x*-ray therapy should be instituted soon after recovery from the surgical intervention. The literature discloses that a number of patients had incomplete operations, since they were operated on for salpingitis; the diagnosis was only subsequently made by the pathologist.

The prognosis is regarded as bad by almost all writers. The mortality from collected statistics averages 40.2 per cent when the removal is complete and 59.8 per cent when it is incomplete. (Bower and Clark.) The recent addition of deep *x*-ray therapy to radical surgery may improve these results somewhat in the future.

REPORT OF CASE

M. O., a single, white Russian woman, aged 32, was first seen on August 31, 1936. She complained of having noticed a "lump in the abdomen" some time previous, although she did not recall exactly when it had first been brought to her attention. Since August 25, 1936, there had been emesis and diarrhea, but pain was not present at any time. Two years previously she had had a similar gastroenteric upset. She had had typhus fever in Russia eighteen years before; a small tumor had been removed from the left breast. Otherwise she had always been well. Her father had died of pneumonia at the age of 50, her mother of heart disease at 48, and one brother died of cancer, the organ involved being unknown. She had three brothers living and well. She had begun to menstruate at the age of 14. Her periods were regular, of the twenty-eight day type, the duration of the periods was seven days, and the flow was considerably more than the normal, although no clots were passed. There was occasional pain, varying in intensity, during the flow, but this was never severe enough to incapacitate her. Her last menstrual period started on August 13 or 14, 1936. Her appetite was poor, her bowels were regular and she had no difficulty at urination. There had been a vaginal discharge of late, but this was stated not to be bloody in character. She had had frequent headaches for the past week; otherwise the history was not remarkable.

The physical examination revealed a well developed and nourished woman, weighing 128 pounds, and having a blood pressure of 160 systolic and 90 diastolic. The head and chest showed no abnormalities. The abdomen contained a tumor which was firm, dull to percussion, immovable and apparently originating from the pelvis. The external genitals and the vagina were normal; the vaginal introitus and the cervix were those of a nulliparous woman and showed no defects. The uterus and the adnexa were involved in a large mass filling the pelvis and rising to the level of the umbilicus. It was impossible to palpate these organs separately. The anus, rectum and urethra were normal. In examining the extremities no abnormalities were discovered.

The urine was normal. The blood count showed the following: hemoglobin (Sahli) 61 per cent; red blood count 3,420,000; white blood count 10,400. The differential count revealed 77 per cent polymorphonuclear leucocytes, 19 per cent lymphocytes and 4 per cent large monocytes. The smear demonstrated anisocytosis, achromia and stippling of some of the red cells.

The patient was referred to the private pavilion of the Beth Israel Hospital, with the pre-operative diagnosis of malignant pelvic tumor. On September 2, 1936, a laparotomy was performed under general anesthesia. After the opening of the abdomen and the inspection of the pelvic contents, the diagnosis of *bilateral*

malignant ovarian cysts with metastasis in the pelvis and small myomata uteri was recorded.

Operation. The operation consisted of a radical abdominal hysterectomy with bilateral salpingo-oöphorectomy.

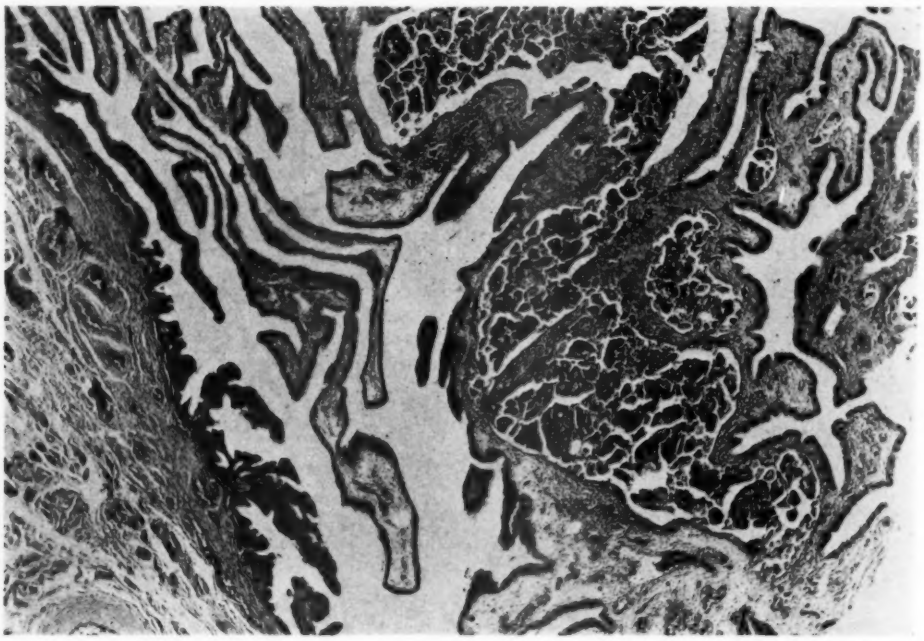

FIG. 1. Left Fallopian tube, low power. Below and to the right, papillary carcinoma growing into the lumen of the tube and replacing the normal epithelium seen to its left. Above, large masses of carcinomatous cells distending the lymphatics of the mucosal folds.

A median pelvic incision starting at the symphysis and extending two inches (5 cm.) above and to the left of the umbilicus was made. The peritoneal cavity was entered between the bellies of the recti muscles. There was found an ovarian cyst the size of a squash which filled the pelvis and had its origin in the left ovary. The right ovary was the seat of an ovarian cyst the size of an orange and a small myoma was discovered in the uterine wall. The tubes were edematous, large, hemorrhagic and firmly adherent to the ovarian masses. The intestines were walled off with moist gauze strips. In order better to expose the deeper structures, the pedicle of the large cyst on the left was cut between clamps and the ovarian tumor was removed. The left broad ligament was opened; it was found that there was a metastasis the size of a lemon encircling the left uterine artery which was enlarged to the size of a lead pencil. The left ureter was dissected for an area of about 3 inches (7.5 cm.) and pushed to the side of the pelvis. The artery was ligated above and below the mass, and the mass itself was removed and sent to the pathologist for a frozen section. On the right the ovary was adherent to the broad ligament, apparently an extension of the malignant process. The ovary was separated from the right broad ligament, the infundibulopelvic and round ligaments on each side were doubly ligated with chromic catgut and cut between the ligature, and a clamp placed mesially. The bladder was freely separated from the uterus and vagina. The right uterine vessels were doubly ligated with chromic catgut and cut, as were the left uterine vessels below the original ligatures. The vagina was opened posteriorly below the cervix and a circular incision was carried around the cervix, thus removing the entire uterus, a vaginal cuff, the metastasis on the left, the two ovarian tumors and the tubes. On palpation, the iliac glands were found to be not enlarged and were therefore not removed. The vagina on each side was closed with a running stitch of chromic catgut, leaving a large central opening through which was introduced a double strip of iodoform gauze, the ends of which were placed over the parametria. The anterior and posterior leaves of peritoneum were united over the gauze by a running suture of No. 1 chromic catgut. The sigmoid was led into the pelvis and the omen-

tum was brought down. The appendix was large, but otherwise appeared to be normal and was not disturbed. The abdominal incision was made on the last date: "The incision is well healed, there is a moderate reaction of the skin to *x*-ray therapy. The vaginal vault is healed

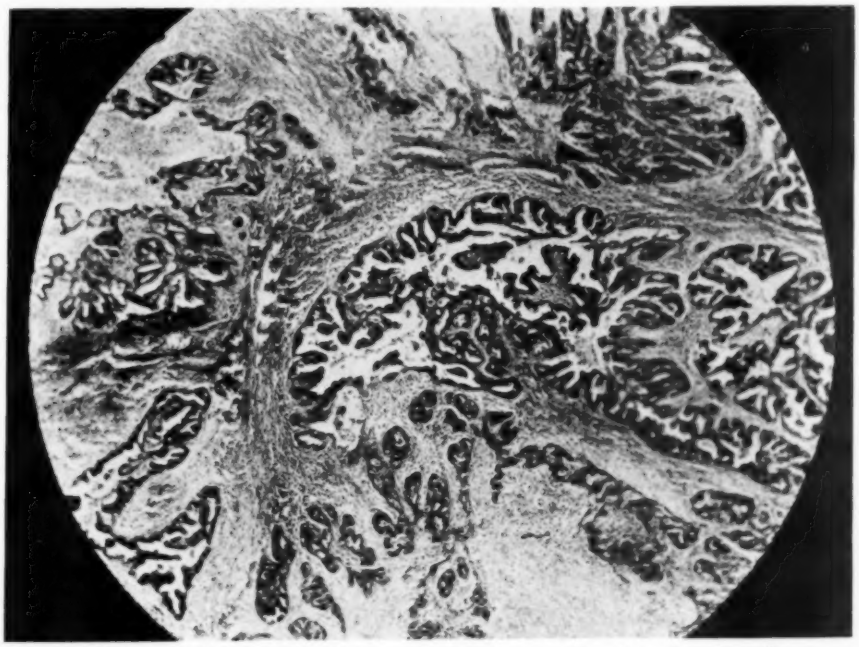

Fig. 2. Right ovarian metastatic tumor, low power. Portion showing the most highly differentiated region in the metastatic masses, with tendency to papillary growth. To the left, more undifferentiated, more invasive areas.

closed in layers. During the operation the pathologist reported that frozen sections made from the metastatic mass on the left showed the presence of a malignant condition.

Convalescence. The patient made a good recovery from anesthesia and her convalescence, although complicated by pyelitis, was satisfactory. The temperature rose to 101 degrees F. (38.3 degrees C.) on the first and fourth post-operative days, and again to 103 degrees F. (39.4 degrees C.) on the fourteenth day. Pyelitis was discovered at this time, but rapidly subsided under the administration of urinary antiseptics and forced fluids. The highest pulse rate was 110 on the third day, while the respirations rose to 24 on the fourth and eighth days. The vaginal iodoform wick was removed on the fourth day and the abdominal sutures on the ninth day; the incision had healed by first intention. The patient was discharged from the hospital September 24, 1936, and was referred to Dr. Harry Friedman for deep *x*-ray therapy.

She was seen at the office on November 2, 16, 30; December 26, 1936, January 23 and February 20, 1937, the following note being and high in the pelvis. The rectal examination shows the parametria to be soft and supple. There is a slight amount of scar tissue on the left but no sign of recurrence.

Thus far, 6200 Roentgens have been administered in thirty-one sessions, this treatment having been completed on November 6, 1936."

Pathologic Report. The material removed at operation was sent to Dr. M. J. Schlesinger, pathologist at the Beth Israel Hospital, who submitted the following report:

The specimen consists of a fusiform mass, labelled A, a large globular mass with tube attached, labelled B, a uterus with tube and ovary attached. The fusiform mass, A, is said to be taken from between the two leaves of the broad ligament on the left side. The mass measures 7 by 3 by 3 cm., and along the length of this mass runs an artery which measures 4 by 0.3 cm. Its lumen, which is lined by a gray wall, is easily patent to a probe. The surface is pink-gray in color. The mass is pink-red on the outside and the surface is lifted up in many places by pea-sized to bean-sized masses. Cut surface reveals a gray surface on which can be seen numerous soft, yellow flecks and inter-

lacing pink-blue strands. There is no regular arrangement of these structures. The surface is somewhat hard and gritty to the cut. The moves as a mass to the knife point and which fades into the surrounding tissue. The cut surface is pink-gray in color with numerous

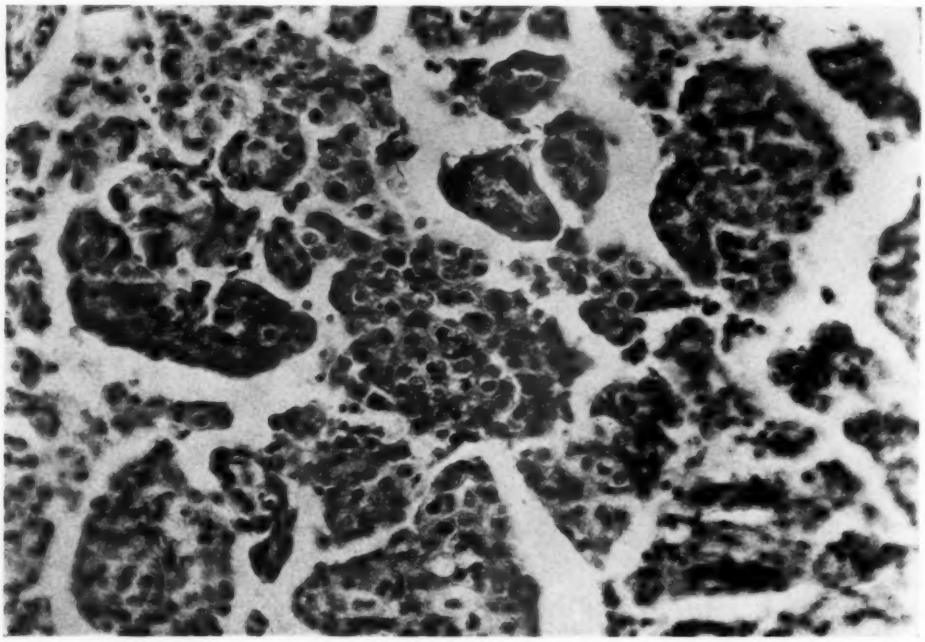

Fig. 3. Right ovarian metastatic tumor, high power. Markedly undifferentiated cells with scant stroma, numerous mitoses, tumor giant cells, and much irregularity of the size of the nuclei.

whole surface moves as one in the gray areas when touched, whereas the yellow areas are very soft to the touch and depressed. In some areas the mass appears to be distinct from the surrounding tissue; in others it fades into them. Frozen section of this mass reveals carcinoma. This specimen is labelled A.

The largest mass is globular in shape, very much distorted by fluid under pressure. It measures 14 cm. in diameter. When punctured, it is found to contain about 700 to 800 c.c. of brown, thin, fluid which contains inspissated, amorphous, light brown collections of tissue. The surface of this mass is attached to the parovarium along one side of which runs the tube, labelled B. The surface is otherwise gray-pink in color and numerous vessels under the surface, which resemble dilated veins, can be seen. In one spot, near the proximal end of the tube, the surface is irregular and red-yellow in color. Adjacent is a larger globular thickening of the wall of the mass; from this, smaller globules project. These smaller globules also project from the wall at varying distances from this larger globule. This largest globule is 3.5 cm. in diameter and the cut surface reveals a tissue which cuts with a gritty sensation, which yellow flecks. On the inside of the large cystic mass one surface of the globular mass, which is pink-gray in color, can be seen. The rest of the inner wall of the cystic mass is brown-green in color, gray in other places, and it is quite irregular, made so by the presence of numerous, variously arranged, amorphous collections of material, such as have been previously described in the fluid of the large cystic mass. From the surface of the globular area arise a few papular excrescences which are firm, green-brown in color and varying from 0.1 cm. to 1 cm. in diameter. The thickness of the greater part of the wall is 1.4 cm. Frozen section of the globular mass in the large cyst reveals carcinoma.

The specimen of the left tube and ovary is labelled B. This consists of a left tube which measures 6 cm. in length and 0.8 cm. in diameter. It is gray-red in color, patent to a probe from the fimbriated extremity to a point 1 cm. from the proximal extremity.

The right ovary is lobulated, forms a large mass, rough, globular in shape, measuring 9 by 7.5 by 6 cm. The surface is made grossly irregular by numerous cystic dilatations which vary in size from 0.8 cm. in diameter to the

largest which measures 6 cm. in diameter. Some of these dilatations are cystic and are blue-gray in color. Others are solid, firm in consistency and yellow-pink in color. Cross section reveals a surface which is partly cystic and partly solid. The solid portion resembles that found in the cross section of the solid mass which made a globular thickening in the wall of the large cystic mass previously described. This section, which is yellow-gray in color with an occasional fleck on it and which fades in the surrounding tissue, cuts with a gritty sensation. The cystic structures contain from a straw to a serosanguineous fluid and the walls are brown-gray in color. On the otherwise smooth surface of the largest cyst can be seen irregular masses of amorphous material.

The right tube measures 5.5 cm. in length and 0.5 cm. in diameter and is patent to a probe to a point 2 cm. from the proximal extremity. The tube is pink-red in color and the distal extremity has on it a cyst of Morgagni.

The uterus, which measures 10.5 by 6 by 4 cm. to 2.5 cm., has been removed by panhysterectomy and is triangular in shape. The external os is small. The mucosa about it is pink-gray in color and smooth. The surface of the uterus is covered by a pink-gray serosa which has numerous puncture marks on it and is lifted up in one area by a pink, bean-shaped area, measuring 0.8 cm. in diameter. Cross section of this shows a pale gray, whorled surface. The uterus is rough along the sides where it is opened. The cervical canal contains a glairy, mucoid material. The endometrium is pale yellow in color and is shaped up in places. The myometrium is of uniform consistency, except in isolated, occasional areas where the resistance is increased by some pea-shaped masses which on cross section reveal a whorled pink-gray surface. The myometrium measures 1 cm. to 2 cm. in thickness. The above three are all labelled C.

Microscopic Examination. Sixteen slides were prepared. Microscopic examination of sections marked A, reveal sections of artery, not unduly thickened and showing no evidence of inflammation.

Sections of the fusiform mass reveal that the background of rather loosely arranged connective tissue is much invaded by an abnormal type of cell. This type of cell is arranged in many sections in clumps with no tendency to papillae formation. In other sections the arrangement is distinctly papilliform. These cells stain darkly. The cell boundaries are relatively indistinct, but the nuclei are very distinct, occupying most of the cell. Numerous mitotic figures and occasionally a giant cell are seen. The cytoplasm in the papillae stains blue. The connective tissue core is very thin. The tumor cells are very much piled up and the papilliform arrangement in some places is very complex. Numerous areas are seen where necrosis of the tumor cells and stroma has taken place. This necrosis is characterized by the presence of a homogeneously staining, gritty material, cellular debris, and nuclear debris. In some places there are polymorphonuclear and round cells. The lymphoid tissue is prominent, with an occasional aggregation.

In sections marked B, the left tube shows numerous blunted, papillary infoldings, lined by relatively normal columnar epithelium in places, but in other places the epithelium appears to be piled up and takes a rather dark stain. The connective tissue core of the papillae is thickened and shows marked round cell infiltration. The lymphatics in the connective tissue core are widely dilated and most of them contain clumps of tumor cells which have no definite arrangement, but are polarized and appear similar to those previously described in the fusiform mass.

Sections of the left ovary, also marked B, reveals one surface which is lined by a connective tissue stroma, containing large cells whose cytoplasm is filled with a tan pigment. The ovarian stroma is invaded by tumor cells which appear in places in the arrangement of clumps. In other places true papillae are formed. In the stroma numerous areas where single tumor cells have invaded the interfibrillary space can be seen.

Examination of sections marked C reveals the presence of endometrium lined by columnar epithelium. Endometrial glands are most abundant, are lined by columnar epithelium, piled up in the form of papillary projections in the lumen. The stroma is composed of small and large round cells. An occasional polymorphonuclear cell is seen.

Sections of tissue composed of very densely arranged whorls of smooth muscle, surrounded by a capsule of flattened smooth muscle, are also studied.

Sections of cervical tissue, covered by squamous stratified epithelium, are studied. The basilar membrane is intact and there are a few round cells in the subepithelial tissue.

Section of right tube, also called c is studied. Papillary infoldings are numerous but somewhat clubbed. They are lined by columnar epithelium. The connective tissue stroma in the core is increased and infiltrated with round cells.

Section of right ovary, marked c, is also studied. This stroma is replaced in many places by tumor cells arranged in clumps, and also in papilliform formation. The collagenous connective tissue can be seen to be invaded by the polarized cells.

Diagnosis. Papillary carcinoma of left Fallopian tube; metastatic carcinoma of both ovaries and left mesosalpinx; hemorrhagic cyst of left ovary; chronic salpingitis; leiomyoma of uterus; hyperplastic endometrium; negative cervix.

SUMMARY

Primary carcinoma of the Fallopian tube is an infrequently found pathologic condition. Up to 1935, Robinson had found but 349 cases in the literature. It frequently, though not always, follows chronic inflammation of the tubes, from which it is usually not differentiated if the tube is removed intact. The diagnosis, as a rule, is established by the pathologist on microscopic examination of the oviduct. The opening of all tubes after their ablation and their inspection for papillary projections, as advised by Gupta and recommended by Kahn and Norris, would not infrequently prove helpful in establishing the diagnosis. The last named authors suggest that, in the presence of a brownish or bloody discharge, not accounted for by a curettage, in a woman beyond 40, tubal carcinoma should be suspected and the oviducts should be carefully palpated for enlarged areas. Because a number of patients have been operated on with the diagnosis of chronic pelvic inflammatory disease, incomplete operations, which are not a satisfactory method of treatment, have been performed. This may account, in part at least, for a number of poor results reported in the literature.

The treatment should consist of a panhysterectomy with bilateral salpingo-oöphorectomy and the excision of palpable glands. The operation should be followed by deep *x*-ray therapy. This form of treatment may improve the results of the future. In my own case, the diagnosis of primary carcinoma of the Fallopian tube was not established even at operation. The ovarian metastases appeared so much more important than the tubal lesion that an operative diagnosis of ovarian carcinoma was made. The histologic examination of the excised pelvic organs revealed the true diagnosis. While the patient has made a good operative recovery and has been subjected to deep *x*-ray therapy, the prognosis should be guarded and only time will reveal the ultimate result.

I am indebted to Dr. M. J. Schlesinger, pathologist at the Beth Israel Hospital for the photomicrographs illustrating this article.

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PITFALLS IN THE HISTORY AND DIAGNOSIS OF FEMALE STERILITY

REPORT OF A CASE, CURED BY SURGERY

ANTHONY WOLLNER, M.D., F.A.C.S.

Assistant Professor of Gynecology, New York Post-Graduate Medical School Hospital of Columbia University

NEW YORK CITY

THE problem of sterility has become complex. Sterile mating with demonstrable fault in one of the partners, while not always curable, at least enables the physician to make a definite diagnosis. On the other hand, those cases in which a thorough investigation of both partners fails to reveal any abnormalities constitute one of the most vexing problems in gynecology. It is well known that sterile marriages, in which partners are apparently normal, are not rare. The gynecologist, after having exhausted all available diagnostic measures, may also utilize the scientific resources of biology, chemistry, pathology, physiology, and endrocrinology, yet in certain cases the cause of sterility will remain obscure.

The following case, which came under my observation, is reported, because it demonstrates certain pitfalls, on the recognition of which may depend proper and successful treatment.

A woman, 30 years of age, came under observation in August 1933. She was apparently in perfect health, her only complaint being sterility. She had menstruated regularly since the age of 14 and had been married for five years. She had had no previous illnesses and never had any symptoms of pelvic distress. A year before consulting me she had been under the care of a gynecologist in another city for the same complaint. Her husband had been found normal, and she had been subjected to repeated pelvic examinations and gas insufflation tests. No cause for the sterility having been found, the patient was advised to wait, with the assurance that a pregnancy probably would occur eventually.

Upon her consultation with me, a thorough investigation of both the patient and her husband was made again. The husband was found to be normal and the gynecologic examination of the patient was negative. The structure of the vagina and vaginal fornix, as well as the position and appearance of the portio, were normal. The uterus was normal in size, in good position, and freely movable; the tubes were not palpable, and both ovaries were of normal size and not tender. Hysterosalpingography was therefore performed after the next menstrual period. The injection of oil was easy, no undue pressure being used to introduce 5 c.c. of oil into the uterine cavity. The picture of the *x*-ray is shown in Figure 1. On superficial inspection, the uterus is seen filled with the opaque medium, one tube is closed at the uterine end, and the other one patent, but there is no evidence of oil in the free pelvic cavity. On closer examination, however, the uterus is found displaced to the left side, deflected inferiorly in its vertical axis. The tube which is visible in the left half of the pelvis is the right tube, and the end seems to be freely mobile in the abdominal cavity. A large amount of oil is present at its fimbriated end. This tube's most distal portion is at the level of the left sacro-iliac joint. The direction of the exuding oil from the fimbriated end indicates that the abdominal opening of the tube is pointing upward. A careful study of the hysterosalpingogram establishes the presence of an anatomic disproportion between the ovaries and the course of the patent tube. After a study of the *x*-ray plate, the patient was again examined bimanually, and both ovaries were distinctly palpable deep in the pelvic cavity. It did not seem probable that the ovum exuded from either of the ovaries could find its way into the patent tube, the fimbriated end of which was seen to be so remote among intestinal coils.

When the situation was explained to the patient she requested that any abnormality should be corrected.

Operation was performed in October, 1933. First a dilatation and curettage were done and a moderate amount of normal looking endometrium removed. The left uterine cornu, apparently the site of an occlusion, was thoroughly scraped with a small, sharp curette. After the curettage, a small, midline incision was made, without undue exposure of the abdominal contents. Inspection of the internal genital organs revealed a small, abnormally movable uterine corpus. The stabilizing apparatus of the uterus was markedly elongated and atonic. The left tube was 8 cm. in length, moderately thickened, and agglutinated at its fimbriated end. The ovary on this side was normal. Only the uterine third of the right tube was visible at first. Bringing this portion into the field, the palpating fingers followed the very thin tube higher up in the abdominal cavity, and its fimbriated end was located near the base of the appendix. This tube was 15 cm long and had an unusually broad mesosalpynx, which allowed it to float freely among the intestinal loops. The right ovary was somewhat larger than the left one, and was fixed by adhesions to the posterior surface of the broad ligament. The adhesions almost completely covered the ovarian surface.

Fig. 1. Left tube closed at the uterine end, right tube patent, displaced.

The right ovary was freed from its adhesions, and after it was brought into view, a small follicle cyst, the size of a peanut was seen on its surface. This portion of the ovary was resected and the rest of the surface scraped to free it from minute adhesions. The mesosalpynx of the right tube was plicated, thereby bringing its fimbriated end closer to the ovarian surface. The sealed fimbriated end of the left tube was easily opened by manipulation with a small probe, and inserted into the tubal lumen. No obstacle was encountered until the tip of the probe reached the uterine cornu, and with gentle pressure and patient manipulation this obstruction was overcome. I believe that the patency of this tube was thus reestablished. The abdomen was closed in layers. The patient made an uneventful recovery, and left the hospital on the twelfth post-operative day.

In May 1934, the patient reported that she had missed a menstrual period. No pelvic examination was made at this time, but the pregnancy test (Aschheim-Zondek) was positive. In June, the patient began to bleed after lifting a heavy object and two days later she expelled the products of conception completely. The bleeding stopped within five days, and five weeks later she resumed her regular menstrual periods.

At the end of November, 1934, the patient again reported a missed period and the pregnancy test was again positive. This gestation was carried to full term. In August 1935, the patient was admitted to the hospital with ruptured membranes, and the vaginal examination revealed one finger dilatation, a thick, rigid cervix and a floating vertex. There were weak contractions every five minutes. After twelve hours of observation, there was practically no progress of labor, and increased bloody show, and the vertex still was floating. The cervix had not dilated further, and manual stretching failed. The fetal heart sounds were of good quality. Considering the importance of a live baby in this case, I did not feel that further temporizing was justified, and a low classical cesarean section was performed. Practically no liquor amnii was present, but a living, seven and one quarter pound female baby was delivered. After closure of the uterine wound the adnexa were inspected. The abdominal end of the right tube was found near the ovarian surface, and the right ovary revealed a true corpus luteum. Apparently the correc-

tion of the tubal displacement was the deciding factor in bringing about the conception. Mother and baby are well at the present time.

COMMENTS

A case of female sterility is presented, in which the application of all usual diagnostic methods did not reveal the true cause of the sterility. Subsequent laparotomy, however, demonstrated an intra-abdominal condition which rendered conception impossible. In the study of this case certain features of the sterility problem were observed which merit further discussion.

In the clinical history of a female sterility case special attention is paid to previous inflammatory processes in the pelvis. In the interpretation of such a history it is wise to remember that the Fallopian tubes possess a tremendous recuperative power. Another case came under my observation, in which an exploratory laparotomy was performed after a criminal abortion with perforation of the uterus. The patient developed grave peritoneal symptoms, and at the operation a large amount of serous fluid was found in the abdominal cavity, the inflamed omentum and intestines were matted together, covering the site of the perforation, and a bilateral adnexal mass enveloped the ovaries. The abdomen was closed with drainage. The patient had a slow and stormy recovery. Three years later the patient became pregnant and carried her pregnancy to term without any complications. This observation demonstrates the capacity of the internal female organs for complete restoration to normal after an extensive inflammatory process. Adnexal inflammations do not justify a diagnosis of permanent sterility. On the other hand, extensive adhesions and tubo-ovarian masses may be found at operation, although the patient cannot recall any previous pelvic inflammation or distress. In the case herein reported, distinct evidence of a previous inflammation was found in the left tube and right ovary, although the patient could not remember any symptoms of such a condition.

To reiterate, the clinical history or evidence of a bilateral adnexal inflammation does not necessarily interfere with subsequent pregnancy, and the absence of clinical symptoms or findings of inflammatory changes in the internal genitals does not exclude the presence of a pathologic condition which makes conception impossible. It is evident therefore, that in every case of sterility, irrespective of the clinical history and bimanual findings, a thorough investigation of the tubes and ovaries is essential.

Hysterosalpingography is one of the most valuable diagnostic methods to determine the condition of the internal female genitals. The *x*-ray visualization of the uterus and tubes gives detailed information that cannot be obtained by the insufflation test. The contour, size and position of the uterus can be determined, the exact site of tubal occlusions is visible, and furthermore, displacements and anatomic peculiarities of the tubes can be recognized by proper interpretation of the pictures. In the case reported here the Rubin test indicated patency, although the subsequent hysterosalpingography demonstrated an abnormally movable and displaced uterus, one closed tube and the patent one markedly displaced. In the interpretation of a salpingogram it is important to scrutinize the pictures for details which may be missed on superficial inspection. The anatomic course of the tubes and the location of the fimbriated ends when correlated with the location of the ovaries, as determined by bimanual examination, may provide a valuable clue in certain cases with regard to the cause of sterility. Dangers attending hysterosalpingography can be avoided by careful selection of the cases and proper technique.

Notwithstanding the great progress made in diagnostic procedures, methods are still lacking by which the most fundamental function in the female, ovulation and the discharged ovum's entrance into the Fallopian tubes, can be ascertained. Sterility due to ovarian dysfunction is not purely

an endocrine problem. The external secretory function of the ovary, the ovulation, may fail because of local pathologic changes in the ovarian cortex. Sclerosis, acquired thickening of the cortex, or adhesions, may interfere in a mechanical way with the normal discharge of the ova. No objective or subjective symptoms indicate such conditions, and no infallible diagnostic procedure is available to discover them. Furthermore, we have just a rudimentary knowledge of the mechanism involved in the entrance of the ovum into the Fallopian tube. We do not know how much importance should be attributed in the determination of the possibility of conception to such conditions as: (1) ovarian or tubal displacements with resulting dislocation of the fimbriated ends from the ovary; (2) congenital or acquired abnormalities of the fimbriae; (3) impaired peristalsis of the tubes; and (4) lack of tonus in the genital apparatus.

We must realize that the available diagnostic armamentarium does not enable us to rule out with absolute certainty the cause of sterility in the female. In those cases in which all investigations yield negative results, an exploratory laparotomy may reveal the cause of the sterility. Inspection of the internal genitals through a small incision, made under local or light general anesthesia, does not subject the patient to undue risk. On the other hand, it does enable the operator to make a definite diagnosis. It will reveal unsuspected abnormalities in certain cases, the correction of which may solve the problem. A typical example of this was observed in my reported case.

Sterile mating is often a serious problem, and the solution is only possible by an accurate diagnosis of its causes. After the utilization of all available diagnostic measures fails to reveal a cause of the sterility, exploratory laparotomy is justified.

SUMMARY

1. A case of female sterility is reported in which the careful reading of the hysterosalpingogram intimated an abnormality as the cause of sterility. Its presence was verified at subsequent operation, and the sterility was cured by simple corrective surgery.

2. A thorough investigation of the internal genital organs is essential in each sterility case, irrespective of the clinical history or bimanual findings.

3. Hysterosalpingography is an indispensable aid in the diagnosis of sterility.

4. When the application of all available diagnostic procedures fails to reveal the cause of sterility, exploratory laparotomy is justified to establish a definite diagnosis.

FOREIGN BODIES IN THE UTERUS

HOWARD C. CLARK, M.D.

Attending Obstetrician and Gynecologist, Wesley, St. Francis, and Sedgwick County Hospitals

WICHITA, KANSAS

DUE to the propaganda of birth-control clinics and lay literature advocating birth-control, many pessaries have been devised for use in the cervical canal and in the uterine cavity. The so-called gold spring pessary is the latest improvement in cervical devices, while the Graefenberg ring is the most popular intra-uterine pessary. Both are presumably for therapeutic purposes, but in reality they are used strictly as contraceptive agents.

Illegitimate practitioners and a large number of none too scrupulous members of the medical profession advocate the use of this supposedly harmless device. However, from this report and from a recent survey of the literature, it appears to be a very dangerous type of contraceptive. Careful search has revealed a small number of papers in the French and German literature, but only a few isolated references in the English and American journals regarding this type of contraceptive. Most of the American authors restricted themselves to a description of individual cases and not to the serious complications that may arise from the so-called harmless pessaries.

It is not my purpose to argue about the number of pregnancies a mother should have. However, I feel that it is the duty of the profession to warn the laity that the wearing of cervical and uterine pessaries is not a harmless and sure preventive of conception, but causes complications of pregnancy and serious pathology of the generative organs.

CASE REPORT

The patient, Mrs. R., aged 26, had always enjoyed perfect health. On August 3, 1935, she had a normal spontaneous delivery. On February 18, 1936, symptoms of burning, frequency, discharge and dyspareunia began to bother her. A smear showed an acute gonorrhea. When she was admitted to the Sedgwick County Hospital February 18, 1936, the patient complained of nausea, bilateral pain, temperature and discharge. The last three menstrual periods had been irregular and painful.

A pelvic examination revealed the perineum in good repair, the cervix fixed, and the uterus normal in size and position. The right tube was enlarged to the size of a lemon. The left tube was the size of a grapefruit and fluctuating.

A diagnosis of suppurative salpingitis with pelvic abscess was made. Since under conservative treatment the patient became progressively worse, it was decided to drain the pelvic abscess. On February 25, 1936, under general anesthesia, a bilateral salpingectomy was performed. The patient made an uneventful recovery and left the hospital in twenty days.

On July 23, 1936, the patient returned to the hospital complaining of afternoon temperature, frequency of urination and severe pain in the right lower quadrant. Vaginal examination showed a uterus normal in size and position, with a mass in the right fornix measuring 6 cm. by 4 cm. A smear was negative and a urinalysis showed many pus cells. Cystoscopy was done on July 25, 1936, and the urologist reported trigonitis, pyelitis and obstruction of the right ureter due to an inflammatory process in the pelvis.

The patient was put on expectant treatment, but did not improve. Cystoscopy was repeated and an *x*-ray was taken of the catheter in the ureter. The roentgenogram showed a metal ring in the pelvis. (Fig. 1.)

The patient then remembered that after her delivery in 1935, she had paid a physician to put a silver ring somewhere to prevent future pregnancies. Her later pelvic trouble had caused her to forget the incident completely until the *x*-ray provided a reminder. She did not want the ring removed, and accordingly the pyelitis continued. In December menorrhagia forced her to come to the hospital again.

On December 16, 1936, with a catheter in the uterus, we rechecked her pelvis and found the ring to be in the cul-de-sac. It had perforated the uterus and was the etiologic factor responsible for both the pelvic abscess and the pyelitis. (Fig. 2.)

Fig. 1. Catheterization of right ureter, showing silver ring in the pelvis. (July 25, 1936.)

Fig. 2. Catheter in uterus, showing ring in the cul-de-sac. (December 16, 1936.)

On December 28, 1936 under general anesthesia a laparotomy was performed. In the right side of the cul-de-sac was a hard, indurated mass the size of a lemon. The ring was in the center of the mass and was dissected out by blunt dissection. (Fig. 3.) An appendectomy was performed and the abdomen closed with no drains. The patient made a rapid recovery and left the hospital in fourteen days free from pain and symptoms of pyelitis.

Discussion. An ideal pessary allows the uterine secretions to flow out even though it prevents the spermatozoa from forcing their way into the uterus. This is mechanically impossible if a gold stem pessary or an intra-uterine Graefenberg ring is used. A foreign body of this type serves as a direct channel for bacteria, which travel from the contaminated vagina to the Fallopian tubes and set up inflammatory processes in the pelvic organs.

Many women have, to be sure, worn a pessary for years without evident harm, while many other women have been left with a chronic salpingitis, which may result in peritonitis and death. The gold stem pessary has relieved many cases of acute anteflexion with stenosis of the cervical canal, but it is a complete or partial failure in our even greater percentage of cases. In dysmenorrhea the relief it gives is often temporary. Even in these cases the pessary should be left in the uterus only for four to six weeks to obtain the best results.

An efficient contraceptive is one that has three characteristics; it must be esthetically unobjectionable to women, harmless, and reliable. Intra-uterine devices are esthetically unobjectionable, but they are both harmful and unreliable. It is physiologically unsound to leave a

pessary in place since it tends to produce counterirritation, resulting in erosion, leucorrhea and endocervicitis; biopsy of the cervix after a pessary has been in place for eighteen months shows all these changes. The foreign body acts also to stimulate the slow rhythmic contractions of the uterus and to force the heavier material into the muscle tissue, producing an ulcer. It is impossible for me to conceive of any material in the uterine body that would not irritate the sensitive endometrium and result in endometritis and parametritis and gradually work its way into the abdominal cavity. This occurred in one of my cases and in seven of Gummerts' cases. Reist has found seventeen deaths caused by perforation of the "harmless" Graefenberg ring.

The presence of the ring does not prevent conception. On the contrary, conception may take place, but a pregnancy will rarely develop to term in the presence of a foreign body (as shown by cervical pack, uterine pack, or uterine abortive pastes). Stefko and Louri reported several cases of damage to the embryo resulting in deformed children, due to the occurrence of pregnancy while the Graefenberg ring was in the uterus.

Gummert (quoted by Warren) collected reports of ninety-two pregnancies during the use of the ring pessary; fourteen went to term, while seventy-eight aborted, ten deaths from septic peritonitis occurred. Cregan reported a case of pregnancy in spite of the Graefenberg ring, but the ring caused a spontaneous abortion, after which the patient developed a streptococcus septicemia and died.

Green-Armytage also reports an infected abortion after nine weeks' gestation; a Graefenberg ring was found in the expelled chorion.

Leunch, a strong advocate of contraception, reported his experiences with the Graefenberg ring in 175 cases. It was expelled spontaneously in thirty-five; there were four full term pregnancies and fifty-five instances in which the ring had to be removed in three to twelve months. Two patients wanted a family, three developed gonorrhea and fifty had menorrhagia, pain and leucorrhea. Leunch concludes that about 45 per cent of his patients are satisfied, and that it would appear the ideal means of contraception if it were harmless and reliable, but unfortunately it is not. He has reason to wonder how long the results will continue satisfactory in the remaining portion of his cases. He believes there are other methods more efficient and much less dangerous to the delicate generative organs.

Fig. 3. Graefenberg ring removed from inflammatory mass in cul-de-sac.

SUMMARY

In spite of the many advocates for the intra-uterine pessary, a review of the recent literature and my own experiences prove that the so-called harmless intra-uterine pessaries are unphysiologic, unreliable and dangerous to life.

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The painful micturition of a cystitis, from whatever cause, comes at the end of micturition, when the inflamed internal surfaces come together, but in the painful micturition associated with appendicitis the pain is often at the beginning, so that a history may be given that the patient holds water as long as possible because of the pain caused when the act is started. From—"Appendicitis" by W. H. Bowen (Macmillan).

SCLERODERMA, KELOID AND SCLEROSIS OF THE BREAST*

REPORT OF A CASE

F. RONCHESE, M.D.

Dermatologist, Rhode Island, C. V. Chapin, and St. Joseph Hospitals, and State Hospital for Mental Diseases

PROVIDENCE, RHODE ISLAND

THE following case of progressive atrophy and sclerosis of the skin with subsequent formation of unusual keloids, of calcium nodules and bone tissue, appears as a very rare combination. The involvement of the breast is also a rare occurrence.

S. M., 51, white, female, born in Finland, resident of this country for thirty-four years, is a housewife on a farm. She was referred to the Tumor Clinic of the Rhode Island Hospital November 13, 1935 for a lump on her left breast of about four years' duration.

A daughter is in a sanitarium for tuberculosis of the spine. The patient had influenza in 1918. A tumor of the uterus was removed seven years ago. She is in menopause since.

Onset of the skin condition was many years ago. The patient states that no fevers, trauma, abscesses, fistulous sinuses, or other skin diseases preceded the present disorder, but that the two main lesions on her body began as dimples and progressed slowly to the present appearance.

On local examination both breasts appeared to be small, flat, firm and movable. The surface was irregular with linear retraction and dimples similar to those on the buttocks of old luetic patients treated in the past years with injections of calomel. (Fig. 1.) The feeling at palpation was of an indurated tissue without a sensation of a definite tumor. No glands were found in the axillae.

On transillumination no shadow was seen. On the left breast just lateral to and below the nipple there was a round cystic projection measuring approximately 2.5 cm. in diameter. On transillumination this cyst cast no shadow. On the shoulders and back there were scattered depressions of various sizes from a dime to a silver dollar, regularly round as if they were made by a pressing machine, or similar to the pitting of an edematous skin under finger pressure. (Fig. 1.) The skin on these depressions had a leather-like consistency.

On the right thigh (Fig. 2) there was a large

FIG. 1. Deep retractions about the breasts and dimples on the back, corresponding to the various stages of sclerosis of the skin.

sunken area 10 by 20 cm. wide, with a raised center, triangular in shape having the appearance of a keloid on top of a scar. The keloid was not, as usually, smooth, light brown and shining, but rough and dull, and deep red-brown. The deep brown discoloration of the center was gradually fading toward the periphery. For several centimeters around the raised center the skin was light brown and mottled.

On the left hip, at the level of the groin, there was a similar lesion showing a red-brown patch, hard, not movable, with an irregular keloidal surface in cord formation as if it were thickened folds of skin. (Fig. 3.) Patches of

* From the Department of Dermatology of the Rhode Island Hospital.

ordinary scleroderma were present on both legs. Large varicose veins were also present in both legs. (Fig. 4.)

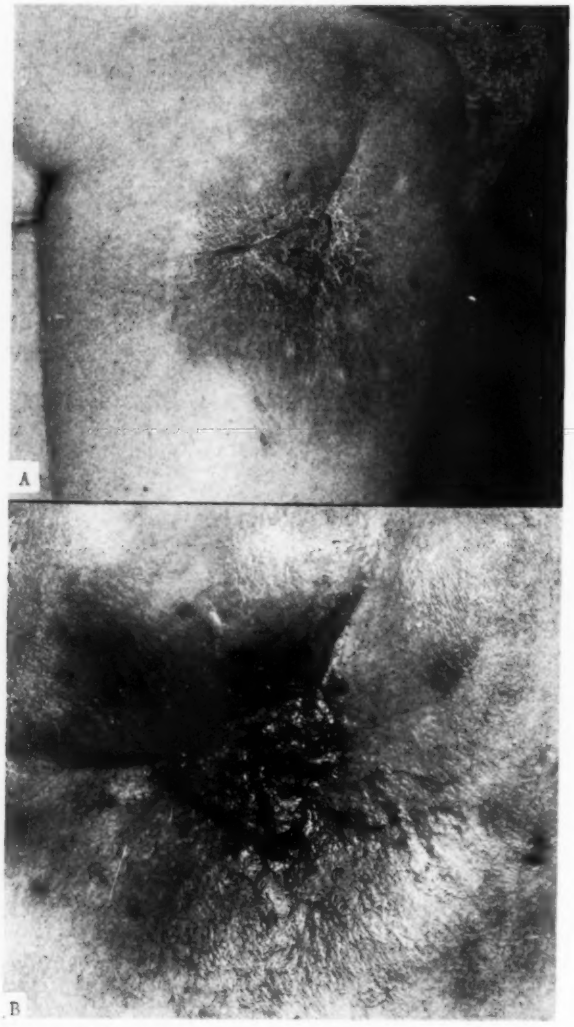

Fig. 2. Advanced stage of sclerosis of the skin and keloid (right thigh). B, two-thirds the actual size of the lesion.

Laboratory Findings. The Wassermann was negative. Hemoglobin was 78 per cent. Blood chemistry showed: glucose 82 mg.; urea nitrogen 5.6; calcium 10.0; phosphorus 3.88; cholesterol 170; total protein 7.8. The urine was normal. The basal metabolic rate was minus 15 the first time and minus 10 a second time. The tuberculin test was negative. *X*-ray of the entire body was negative, although a few miliary calcium nodules were later found in the breast and large calcium nodules and bone formation in the skin.

Operation. A bilateral mastectomy (right radical, left simple) was performed on January 10, 1936 by Dr. P. P. Chase. The recovery was uneventful.

Pathology. Macroscopically (Figs. 1 and 5) the left breast shows a cyst to the left of the nipple which measures 3 cm. in diameter. The cyst is filled with dark amber fluid and attached to its wall is a gray area 2 cm. in diameter, which, on section, is slightly granular and sends fibrous bands into the surrounding fat. There are several fibrous areas through the breast tissue which are firm, elastic and smooth on section. There is also a rounded, encapsulated fatty tumor near the skin surface measuring 2 cm. in diameter. The right breast shows a linear depression of the skin medial and inferior to the nipple, measuring 3 cm. in length. In the breast tissue just beneath this depression is a hard, gray mass, measuring about 2 cm. in diameter, and which on section is granular in areas. The mass sends gray bands into the

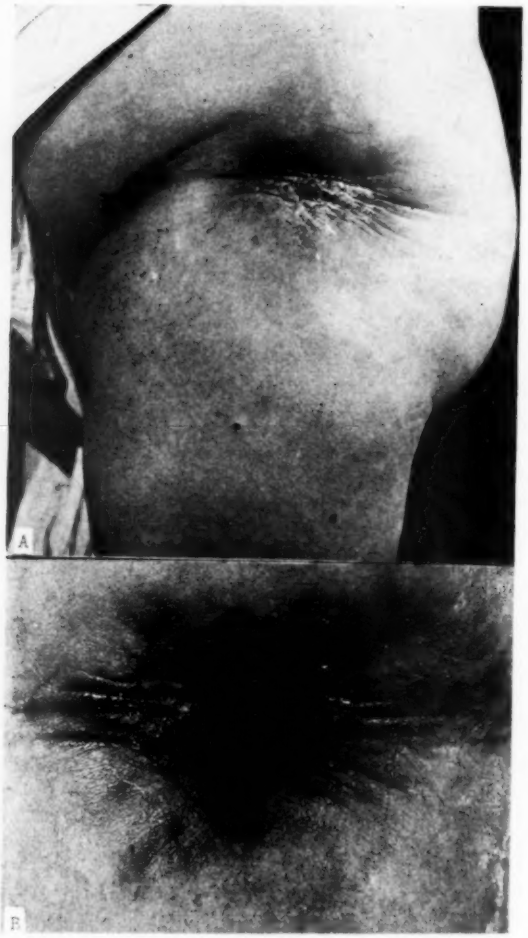

Fig. 3. Area of sclerosis of the skin with linear fold-like keloidal features (left hip). B, two-thirds the actual size of the lesion.

surrounding fat. There is calcification in the center of the mass.

Microscopically, the ductal and acinar epithelium shows marked hyperplasia and a marked tendency to form papillary growth in the dilated ducts and acini. In other places ducts are filled with solid masses of cells. The connective tissue stroma is very dense and hyaline and contains many large accumulations of lymphocytes. The sections of the skin (Fig. 7) show considerable pigment in the basal layer of the epithelium. The papillae are elongated. The dermis and subcutaneous tissue are made up of dense hyaline connective tissue fibers containing many lymphocytes, mostly perivascular. The upper part of the lesion on the right thigh (Fig. 7A), shows the usual structure of a keloid. Hair follicles, sebaceous and coil glands are absent. Further below (B), there are large calcified nodules surrounded by bone tissue. In other sections, from a dimple on the back (C), and from the skin of the breast (D), a transition is clearly visible from a wavy epidermis to a flat and thinned one corresponding to the areas of an early sclerosis (back) and of an advanced one (breast).

Fig. 4. Scleroderma in patches of the usual type, and varicose veins.

Diagnosis. Scleroderma and keloid. Intraductal papilloma of the breast.

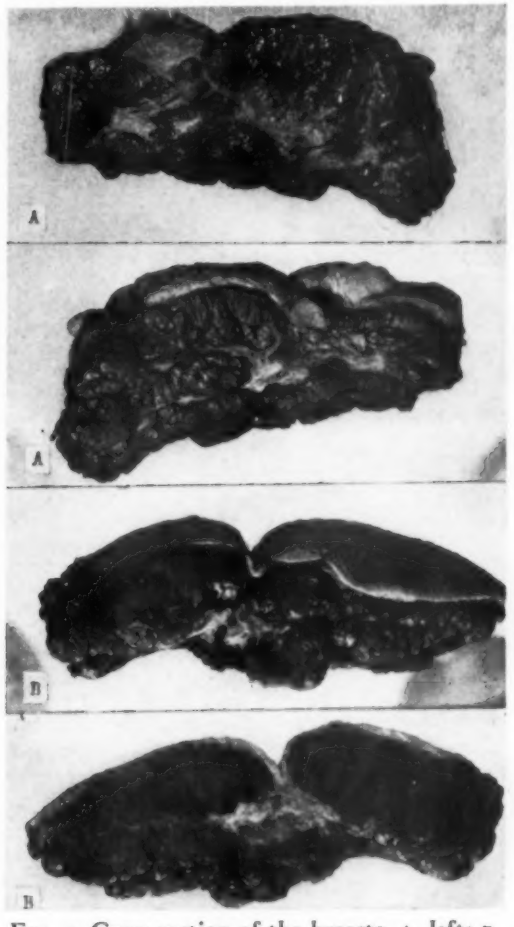

Fig. 5. Cross section of the breasts. A, left; B, right.

Therapy. On reports of a favorable effect of pituitrin on scleroderma, fifty 1 c.c. vials of surgical pituitrin were administered subcutis about every other day. The patient many times complained of abdominal pain after the injection. No improvement of the skin condition was noticed and the treatment was discontinued.

The patient was seen February 26, 1936. Keloids of the usual type developed on all the mastectomy scars. (Fig. 6.) Where a generous biopsy was taken, from the lesion of the right thigh, and where the toughness of the skin prevented the use of sutures, the gap was filled with a similar keloidal tissue so that one could hardly notice that a biopsy had been taken.

The patient was seen again on March 25, 1936 and July 15, 1936, and the keloids on the

operative scar were of the same size as six months previously. The patient showed varicose ulcers on both legs.

FIG. 6. Keloids of the usual type following mastectomy.

COMMENT

Scleroderma is a well known disease involving a large part or the entire body (diffuse scleroderma) or limited to portions of the skin (circumscribed scleroderma, morphea). In both varieties the involved skin has the appearance and consistency of parchment or of heavy leather and it is usually smooth.

In a very few cases nodular or like formations have been described, briefly and more or less clearly, on or with the patch of scleroderma.[1]

Association of scleroderma and keloid is not mentioned in two extensive discussions held by the French Society of Dermatology, one on Scleroderma in 1929, and one on Keloid in 1931. The same can be said for many other comprehensive papers studying scleroderma, dermatomyositis, calcinosis, osteoma of the skin and the atrophodermias.

Of the American textbooks of dermatology only Stelwagon[2] mentions sunken scars and formation of keloids. European textbooks practically all mention morphea tuberosa, but with just a few lines. For instance, Weissembach and Bash[3] outline the rarity of the condition as follows: " . . . sometimes the surface of the patch is still more different from the usual, it is irregular, it is formed of a conglomeration of tuberosities, recalling the appearance of some keloids and making a rare variety, the "morphée tubéreuse."

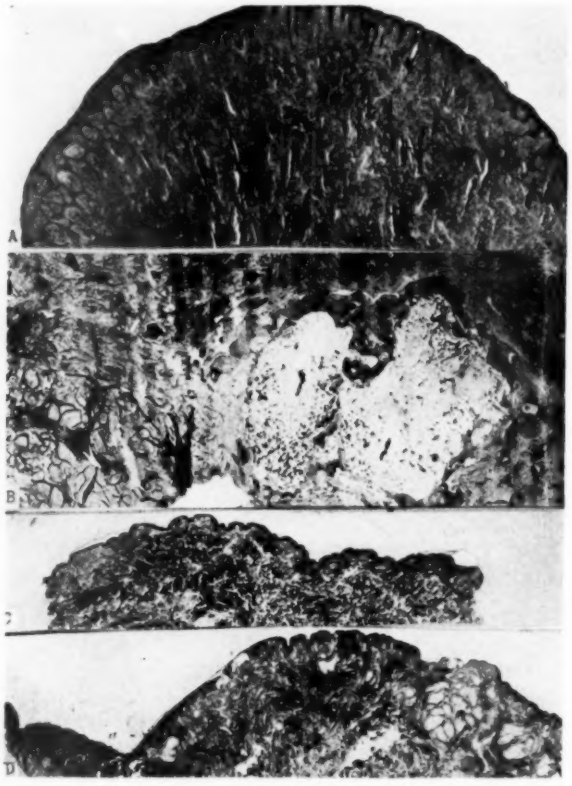

FIG. 7. Low power photomicrograph showing: A, nodule from the lesion on the thigh shown in Figure 2, with typical structure of a keloid. B, an area of calcification surrounded by bone tissue located about 1 cm. below the keloid. C, section through a dimple on the back, as shown in Figure 1, with a flat epidermis corresponding to the area of early sclerosis of the skin. D, section through the skin of the breast with epidermis changing from wavy to flat and thin, and indentations corresponding to the retractions shown in Figure 1.

Nodular formations, called fibromas or fibroma-like tumors, some of them elongated like a cord, and a variety of other tumors were described in cases of acrodermatitis chronica atrophicans showing scleroderma-like features. A clear cut separation between the various atrophies of the skin and scleroderma is often difficult. Scleroderma is often spoken of as a variant, or as the end-result of an atrophic process. However, atrophy has been found to follow resolution of scleroderma in the case of Crawford.[1]

The subject is exhaustively reviewed and discussed by Sweitzer and Layman[4] and by Bertaccini.[5]

In the present case there was not the slightest suggestion of acrodermatitis chronica atrophicans and the nodular formations were absolutely different from those described in this disease. Lichen sclerosus (white spot disease, lichen albus, morphea guttata, morphea plana) was also easily ruled out.

The case was unusual for the peculiar form of sclerosis of the skin showed in its various stages from an hardly visible depression or dimple to a sunken, radiated, scar-like lesion with appearance, consistency and pathology of a keloid.

The keloid in the present case was clinically totally different from the usual type, showing a rough and dull surface and a deep red-brown discoloration. Keloids of the usual type have raised from a biopsy in cases of scleroderma. In the present case the same patient showed two distinct kinds of keloids: a usual type on the surgical scars of the mastectomy (Fig. 6); an unusual one, rough, dull, red-brown, where a biopsy was done on the lesion on the thigh. (Fig. 2.)

The first impression given by the lesions on the thighs was one of an ordinary scar followed by a keloid. No appreciable injury caused it and early non-traumatic lesions were well represented by the dimples on the back. Fairly typical patches of common scleroderma on the legs helped a great deal in the diagnosis.

The beginning of the lesions as a round depression of the skin surface, on inspection, ressembled the case of atrophy of the fatty layer of the skin described by Gilchrist and Ketron[6] or what goes under the name of relapsing febrile nodular non-suppurative panniculitis (Weber-Christian disease) and also the rare form of atrophodermia described by Pasini in 1923[7] and recently by Pierini and Vivoli.[8] But palpation revealed definite sclerosis, completely absent in the cases of Pasini's atrophodermias, and the evolution showed sclerosis and keloid. Furthermore, Gilchrist's and Ketron's patient was seen only a few years ago for another skin condition[9] and no trace was noticed of the previous atrophodermia. In my case, it is very unlikely that any restitutio ad integrum will take place in the future, and the Weber-Christian syndrome is unlikely, since there is no history of fever.

Another peculiar feature of the case, of surgical interest, was the involvement of the breast. Deep dimples, retraction of the nipple, and hard lumps could easily be suggestive of malignancy. In the differential diagnosis of a breast condition, with atrophy and scleroderma elsewhere, sclerosis of the breast should be considered and a surgical intervention might possibly be avoided.[10]

SUMMARY

A case of scleroderma and keloid in its various stages of evolution is described.

It showed patches of circumscribed, progressive atrophy and sclerosis of the skin, with calcinosis and osteosis, having the appearance, consistency and pathology of scleroderma and keloid.

This condition seems to be very rare. It may be similar to what is described, in European literature, as "morphea tuberosa."

The involvement of the breast was of particular surgical interest because it was suggestive of malignancy. The etiology was obscure.

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MIXED TUMORS OF THE PAROTID TYPE

WITH A REPORT OF THREE CASES

PHILLIPS L. BOYD, M.D., F.A.C.S.

Massachusetts Memorial Hospital; Boston University School of Medicine

BOSTON, MASSACHUSETTS

MIXED tumors of the parotid type are of great interest because of their histology, physiology, and their response to therapy. A survey of the present status of these tumors is here presented.

A brief resumé of the history of the pathologic differentiation and classification of these tumors is necessary to appreciate the interesting problems they present. Billroth, Heineke, and Volkmann first described them as endotheliomas, and then later as cylindromas. Krompecker thought of them as basal cell tumors. Marchand, Wilms and others now regard them as composite tumors in which the epithelium plays the most important part, and trace them to an origin analogous to that of the teratoma, except that the embryonic rudiment is separated at a later stage. According to McFarland, sequestration of embryonal cells is the most satisfactory explanation. However, the question as to the origin of these cells from embryonic pharyngeal pouches, gill clefts or buccal remnants has not been answered. It is only within the last twenty years that the cognomen "Mixed Tumors of the Parotid" has reached any prevalence. It must be borne in mind that these neoplasms bear no necessary relation to parotid gland cells, but have been named because of their usual occurrence in the parotid region. The term denotes a group, but, contrary to the usual practice in the classification of tumors, does not describe the basic cell.

Etiology. As has been indicated above, the origin of these cells is in doubt. It is probable that most are congenital and are due to embryonic rests rather than degenerative or entoplastic tendencies of normal structures in the post-embryonic stage of development. Ewing states that there is no single source of mixed tumor. Some of them are adenomatous and may arise from the ducts and acini of the parotid, others are encapsulated and take the form of a basal cell epithelioma. Trauma probably plays no part in the etiology.

Incidence. These tumors are not uncommon. To judge their relative frequency, I quote the following series of cases: in the Mayo Clinic from 1912 to 1917, 103 cases were reported; in the Memorial Hospital in New York, from 1918 to 1921, fifty-nine cases were seen; 0.1 per cent of 6600 cases treated in the Institute for Malignant Diseases in Buffalo were so diagnosed; in the Massachusetts Memorial Hospitals there have been recorded 10 cases prior to those reported here since 1924. In all, 450 cases have been reported in the literature.

Mixed parotid tumors appear most frequently from twenty to thirty, but may occur at any age, and are seen with equal frequency in both sexes.

Symptomatology. These growths are characterized by their lack of pain and by their slow steady enlargement. The patient or his friends usually notice a small mass, rarely smaller than a lima bean, which lies in the region of the parotid gland. Occasionally the other salivary glands, rarely the lacrimal glands or buccal cavity, may be the site of origin. The sites of ten mixed tumors recorded in this hospital are: parotid region, five; cervical region, one; face, two; nose, one; over the mandible, one. There may be a history of trauma, but it is probable that the trauma

has directed the patient's attention to the region and to a previously unnoticed growth, rather than that it has initiated the growth. The small tumor may stay as first noticed for three to five years and then begin to enlarge rapidly. It is when this latter happening occurs that the subject may become alarmed over the blemish to his facial symmetry or he may begin to worry over the possibilities of a cancer or other malignant tumor. Pain may be present, due to pressure of the enlarging growth upon the facial nerve as it decussates through the gland.

The tumors range in size from that of a lima bean to that of a baby's head. The rate of growth is usually slow and steady. According to McFarland, for the growth to attain the size of a walnut usually takes about five years; to attain the size of a lemon, ten years; and to become larger than this, twenty or more years. Occasionally they may become very large within a year. However, unless the pathologic picture resembles carcinoma, the prognosis is no worse than the more slowly growing types as shown in McFarland's series of 130 cases with follow-up study.

Physical Findings. The mass is usually regular, often lobulated, firm, occasionally soft or even fluctuant, non-tender, fixed to the deep tissues but unattached to the skin, and in the near vicinity of the parotid.

Physiologic Characteristics. While there is a strong tendency to recurrence following removal (this occurs in 25–30 per cent of the cases), there is a very slight tendency to metastasize. These tumors are usually considered by most writers as benign, although the microscopic picture is often consistent with a very malignant process. In one series of 90 cases, one had metastasized, and in a series of 130 cases, none had metastasized. Burrows and Wakely believe that cases of mixed tumor of the parotid should be considered potentially malignant. Death may result upon recurrence, due to gangrene, sepsis and impaired nutrition, but very rarely by a generalized spread with the typical exodus of malignancy.

Differential Diagnosis. The differential diagnosis must be made from tertiary lues, tuberculosis, chronic adenitis due to oral and aural sepsis, actinomycosis, sarcoma, carcinoma and other rare adenomas of the face and neck.

Pathologic Characteristics. The gross and microscopic appearance varies considerably. Bland Sutton states that mixed tumors of the salivary glands are pathologic puzzles and the source of much unsatisfactory speculation. They are the most common tumors of the parotid. They are for the most part encapsulated with a very thin membrane which covers all extending strands of cells. They grow by direct extension and compress structures before them rather than invade them. They are firm, elastic, lobulated, often surrounding and compressing the facial nerve, and they may cause a facial palsy if this pressure becomes too great. The color varies from a pale yellow to a pearly bluish hue. The consistency may be mucoid, hard and cartilaginous, fibrous, and even osseous. Palpation may at times give the impression of a lipoma, and at other times of a cyst.

Microscopically the structure is a mixture of epithelial cells of varying characteristics and a stroma of fibrous cells. The following characteristics, given by Boyd as the principal structures may be found all in one specimen: (1) broad or narrow strands of epithelial cells derived from glandular alveoli (these cells may be flattened and arranged in alveolar form); (2) anastomosing cords of small dark epithelial cells with hyperchromatic nuclei; (3) mucoid connective tissue; (4) areas of lymphoid tissue. Any of these types may predominate, and in addition, osteogenic structures may be found. Sometimes the cells are actively secreting. From few to many mitotic figures may be present. Many of the growths are well vascularized and from all microscopic criteria the tumors should be classed as malignant.

However, as has been stated they do not carry the prognosis of a malignant growth.

Treatment. The consensus of opinion advocates surgical excision followed by radium, radon seed or Roentgen ray therapy. However, McFarland seeks to discourage *early* operation as hazardous, since facial palsy due to nerve injury may result, or there may be more resistant recurrences and sepsis. He cites three cases of nerve injury. Other series have shown the incidence of nerve palsy to reach as high as 30 per cent in operated cases, but this latter figure seems inexcusable. The incidence of recurrence in small tumors runs close to 30 per cent with a mortality traceable to the condition of 6.6 per cent. In growths larger than a lemon, the rate of recurrence is less than 15 per cent. In a series of ninety cases, unclassified as to size, nineteen had recurrence with two deaths. Operation tends to cause increased activity in the recurrence.

Simple removal of the encapsulated tumor has permanently cured 70 to 75 per cent of patients. With a transverse incision and with careful blunt dissection, facial palsy should be practically nil. In the recurrences, secondary operation usually cures a good percentage; in 19 cases of recurrence, it was impossible to eradicate the tumor in only three. In the small series at the Massachusetts Memorial Hospitals, no cases of facial palsy have occurred. In the unencapsulated tumors simple surgery is less successful. Radiotherapy, advocated by many roentgenologists, has yet to prove its value as a primary form of treatment; at least so far the results do not equal those obtained by surgery. Benedict and Meigs state that the chief value of radiation treatment is in recurrences and for palliative treatment in advanced cases, but many of their patients represented malignant cases and did not have typical mixed tumors. The Manchester Radium Institute finds from experience that combined operation and radiation is the method of choice, but that interstitial radiation seems to be quite as good. Their cases are listed as follows:

Treatment	Cases	Cures
Surgery and post-operative radiation	8	8
Interstitial radiation	5	4
External radiation alone	3	1

Merritt feels that the combined treatment is superfluous and that the treatment is essentially radiologic. He found that small encapsulated tumors responded to radium seed or needle implants as readily as to excision. Quick and Johnson feel if the growth is favorable for surgery it is likewise favorable for radium. Most radiotherapists, however, agree that primary radiation has little influence on the growths. The combined operation is favored as the method of choice for two main reasons: first, and most important, absolute diagnosis can be made only with the microscope and to do a biopsy and open into the capsule would seem to add to the risk of recurrence, so that primary total excision would be more advisable; second, 70 to 75 per cent of cases are cured by surgery alone, and post-operative radiation raises this to nearly 100 per cent of cures.

I now report three cases treated on the service of Drs. Harvey, Barton and Boyd, to illustrate the advantages of surgery as the primary method of choice.

Case 1. C. D. L., a 14 year old school girl, entered on January 16, 1934, with a chief complaint of a swelling in front of the left ear of four years' duration. This had first been noticed when the mass was about the size of a lima bean. Since this time it had grown slowly and painlessly. She sought medical advice because the lump was becoming noticeable to others. Physical examination revealed an almond-sized tumor mass lying at the lower pole of the left parotid gland and freely movable under the skin. The mass was questionably fixed to the underlying deep tissues.

Under gas oxygen ether anesthesia a friable tumor with a thin capsule was removed in several pieces. The wound was closed without drainage and healed by first intention without any nerve injury. The patient was put through a course of *x*-ray therapy after operation. So far

there has been no evidence of recurrence (June, 1935).

Pathologic Report. The gross examination showed several masses of tissue mottled gray and reddish brown, firm, irregular and glistening. The cut surface was also gray, smooth and glistening. Microscopic analysis was as follows: Section consisted largely of dense hyalinized or loosely arranged myxedematous stroma of a moderately vascular nature in which were occasional discrete groups of small hyperchromatic epithelial cells, tending to be arranged as glands. A few mitotic figures were present. The diagnosis was a mixed tumor of parotid type.

Case II. V. B., a 26-year old housewife, entered February 9, 1934, with a chief complaint of a swelling of three years' duration on her neck below the ear. Three years ago the patient was struck sharply below the angle of the right jaw, and the area became swollen and painful. She was seen by her physician, who advised ice packs, but in spite of this, the swelling did not wholly subside. It had gradually enlarged until six months ago, when it began to enlarge at a faster rate. There was no pain, but the mass was tender on firm pressure. In 1932, operation was advised, but deferred.

Physical examination showed a semi-firm, lobulated mass lying below the angle of the right mandible. The mass was freely movable under the skin and over the underlying tissues, and fluctuated on palpation. Under gas oxygen ether anesthesia a well encapsulated, lobulated tumor was removed. Some gelatinous material oozed from the tumor during removal. The wound was closed without drainage and healed by first intention. No facial palsy or other apparent nerve injury was noted. The patient was referred for *x*-ray therapy, but failed to return. She was two and one-half months pregnant. Even without the *x*-ray therapy, so far there has been no evidence of recurrence (November, 1937).

Pathologic Report. Gross examination disclosed a tumor mass $4.5 \times 2.5 \times 2.5$ cm. in size, mottled gray and bright red, nodular, soft, enclosed by a gray, firm translucent, loosely adherent capsule. The cut surface was yellowish gray, smooth, glistening, and moistened with a gelatinous material. Microscopic study showed a loosely arranged, faintly staining acidophilic myxedematous-appearing stroma in which were large numbers of small, rather hyperchromatic epithelial cells growing in solid sheets and elongated cords and occasionally attempting alveolar arrangement. These cells varied in size, shape and staining reaction, and presented rare mitotic figures. The diagnosis was a mixed tumor of parotid type.

Case III. A. C., aged 76, entered through the O. P. D. in April, 1934, with a chief complaint of a painless swelling below the right ear, of four years' duration. Fourteen years previously he had had a gland removed at the same site because of enlargement. The diagnosis was unknown to the patient. For ten years he had noted no sign of recurrence. However, four years ago a small mass appeared, and enlarged slowly and steadily. It was neither painful nor tender, but the patient felt that his hearing was impaired by it.

Physical examination revealed a mass with three distinct lobes below the right ear. Under novocaine infiltration anesthesia the two lower lobes of the growth were removed for biopsy. Due to the fact that the remaining portion of the tumor surrounded the facial nerve, it was not removed. The pre-operative and operative diagnosis was mixed tumor of the parotid type, probably a recurrence. However, the pathologic report did not substantiate this diagnosis. The tumor was a mixed tumor, but not of the parotid type. The patient was referred for *x*-ray treatment.

Patbologic Report. The specimen was a gray, soft, glistening tumor mass which on microscopic examination was found to consist entirely of small fusiform brightly staining acidophilic cells, tending to have elongated nuclei almost completely occupying the cytoplasm. The characteristic feature was the presence of large numbers of clear spaces filled with a thin basophilic staining, mucinous appearing material. The cells mentioned above tended to be arranged in a peripheral whorl-like mass about these spaces and in some places seemed to radiate definitely from them. The individua cells were quite uniform in size, shape and staining reaction and very rare mitotic figures could be seen. The diagnosis was hemangioendothelioblastoma, probably a low grade malignant growth.

After six months, the tumor mass had not enlarged and gave no symptoms; general health was good except for hypertension. Radiation had been discontinued. The patient died in

December, 1934, of a cause unrelated to the tumor.

SUMMARY

A general resumé is given of the present day knowledge concerning mixed tumor of the parotid type, emphasizing symptomatology, behavior, pathology and treatment, with a consideration of two cases so diagnosed. A third case, which closely paralleled the symptoms and physical findings, is discussed to illustrate how essential it is to be sure of the diagnosis by microscopic means before therapy is started or prognosis given.

The best procedure to follow in suspected cases of parotid type mixed tumors is to excise the mass in capsula, make a positive diagnosis by microscopy, and post-operatively to radiate the site.

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Ludwig's angina usually follows an infection of the mouth. Hence the inflammation is generally confined to the subcutaneous tissues of the upper neck, but the whole of the neck may be affected and even continue to the upper chest.

From—"Surgical Pathology of the Diseases of the Neck" by Arthur E. Hertzler (Lippincott).

MULTIPLE BULLET PERFORATIONS OF SMALL INTESTINES*

TWO RESECTIONS WITH END-TO-END ANASTOMOSES

PAUL K. SAUER, M.D.

Associate Surgeon, Lenox Hill Hospital; Visiting Surgeon, City Hospital

NEW YORK CITY

F A., aged 27 years, a Filipino, was admitted to the surgical service of City Hospital at 2:00 A.M., on August 21, 1936, with a history of having been shot in the abdomen during an altercation. He was first seen half an hour later, at which time he was in mild shock. Treatment for shock was instituted by the house surgeon immediately upon admission. The temperature was normal, the pulse rate 100. The abdomen was tender and comparatively rigid throughout. An entrance bullet wound was found in the left hypochondrium at the left edge of the rectus muscle, and an exit wound in the left lumbar region. There was oozing from both wounds, but no free bleeding. A second entrance and exit wound (whether from the same bullet or not was not ascertainable) was found at the lower end of the right forearm. A basswood splint was applied and was left in place temporarily. A roentgenogram of the forearm showed a badly comminuted fracture at the distal end of the right radius with dorsal angulation at the site of fracture.

By 3:30 A.M. the patient seemed to have recovered sufficiently from his shock to warrant the indicated laparotomy. The pulse quality was good.

Under general anesthesia an incision was made up and down from the entrance wound of the bullet. Projecting through the fascia a piece of omentum was found plugging the hole. The muscle was retracted mesially and the peritoneum entered. Much blood and many clots were evacuated. Careful inspection with as little manipulation as possible revealed that the transverse and descending colon were not injured, but the bullet had passed through the mesentery of the descending colon. No spilled intestinal contents were discovered. During the manipulation just described a perforation was seen in a loop of small intestine. Further examination revealed eleven more perforations in 30 inches of gut. Two perforations were distant enough from each other (3 inches) to permit suture in situ, without constricting the lumen. Beginning about 4 inches above the upper one of the closed perforations was a piece of intestine perforated four times, with the openings so situated that it was thought inadvisable to close them. A quick resection with end-to-end anastomosis was done. No other injury could be found in any proximal loops. About 6 inches below the lower sutured perforation, six perforations were found in a loop about 10 to 12 inches in length. This loop was resected, also, with an end-to-end anastomosis. Bleeding seemed to have stopped, and further search in this area revealed no other injuries. An interesting finding in this case was the total absence of intestinal contents at the time of resection of the two loops. The abdomen was closed in layers without drainage. Except for a mild state of shock twelve hours later, recovery was uneventful, with the highest post-operative temperature 101.2 F. The patient was out of bed on the twelfth day. The radius was treated with skilful neglect with a fortunate result.

The case is presented to show: (1) a patient in whom one bullet caused twelve perforations of the small intestine within a distance of 30 inches; (2) the importance of early operation before the development of symptoms of peritonitis; and (3) that the treatment of shock is always the first consideration. Rushing a patient to the operating room is fraught with grave danger, and in a series of such cases will give an undesirable mortality rate.

DISCUSSION

Dr. Ralph Colp of New York cited the case of a double intestinal resection that he reported in the *Military Surgeon*, February, 1922. This was an individual who had been shot in a gun fight and was admitted to Beekman Street Hospital. At

* Presented before the New York Surgical Society, November 11, 1936.

the time of admission he presented a wound of entrance 3½ inches below the costal margin in the middle portion of the right rectus, which was exuding a foul-smelling, fecal fluid. Operation revealed an intestinal loop which contained four perforations. This was resected with an end-to-end anastomosis. Then, 3 feet farther down, he presented a through-and-through perforation, and because of the fact that single sutures would have compromised the lumen of the intestine, this, too, was resected with end-to-end suture. About one foot from the ileocecal junction he presented two other perforations, which were closed with simple purse string sutures. The patient had a stormy convalescence and on the ninth day presented some pain to the left of his anus. An abscess developed there, and incision yielded some foul smelling pus and one bullet. At that time Doctor Colp was very much interested in reviewing the cases of multiple intestinal resection, and found that from 1900 to 1920 fifty-four cases had been reported. Auray, in the *Presse Médicale* in 1915, reported a case of multiple intestinal resections in which he had performed five separate intestinal resections with end-to-end sutures. Hunt, in the *Boston Medical and Surgical Journal* in 1920, reported a case of triple intestinal resection in which he used three Murphy buttons and the patient recovered. In addition, Kohlman in the *New Orleans Medical Journal* reported a case in which he had done a triple intestinal resection. Up to that time some twenty double intestinal resections had been reported.

The issue is whether to do multiple resections, simply close the perforations, or combine both measures. Where there are only a few feet intervening it might be safer to do a single resection and include all the perforations. Several interesting cases have been reported, but one by Lyons is particularly worthy of note; in his case there were twenty-four perforations and he successfully resected nine feet of intestine. Primarily, one must be guided by the number of perforations and whether one can close them without compromising the lumen of the gut; secondly, whether the blood supply in the mesentery is destroyed so that there might be secondary necrosis of the bowel; and, thirdly, whether the interval between the perforations is such that one can do a single resection safely.

Dr. Charles Gordon Heyd, of New York, inquired whether the author closed his perforations followed by resection as he came upon the perforations, whether he had exposed all the perforations before starting his plastic or anastomotic surgery, or, whether he first did a resection and then went on to another portion of the bowel. That is, were all the perforations known before the first resection was started? They were all known.

A great many patients with multiple wounds were encountered during the war, and as the mortality rose with each resection, it became the custom to expose all the perforations, clamping them off with rubber resection clamps, and then doing one resection. This was followed by rather remarkable improvement in mortality. It may be therefore that in view of all the circumstances in the author's case, it might not have been better to have sacrificed a few feet of bowel in order to do only one resection.

Dr. Sauer, in answer to Dr. Heyd, said that he contemplated doing one resection but feared an inadequate blood supply. A small piece could have been taken out easily, but if a long 30 inch piece had been resected how would the ends have come together? Moreover, the procedure could be done as quickly with two resections instead of a single one.

OSTEOCHONDRITIS DISSECANS INVOLVING THE ENTIRE ARTICULATING SURFACE OF THE DISTAL END OF THE FEMUR

IRVIN BALENSWEIG, M.D.

Cornell Medical College and New York Hospital

NEW YORK CITY

OSTEOCHONDRITIS dissecans is an affliction in which a fragment of articular cartilage, with or without bone nucleus, becomes partly or completely detached from the underlying bone. It invariably arises from the articular surface of joints, the most frequently affected being the larger joints of the body. The lesion may on occasion be bilateral.

According to Mercer,[1] the typical sites in the various joints are as follows:

1. In the knee, the medial condyle.
2. In the elbow, the capitellum.
3. In the ankle, the trochlear surface of the astragalus.
4. In the hip, the superior surface of the head of the femur.

The writer[2] has previously described an instance of osteochondritis dissecans arising from the external condyle of the femur, a type of lesion which is extremely rare. Of the various joints enumerated, the knee joint is by far the most frequently involved. The case here presented is an instance of osteochondritis dissecans involving practically the entire articulating surface of the distal end of the femur.

Etiology. Several theories have been brought forward, particularly those of traumatism and aseptic necrosis resulting from thrombosis of the blood supply to the part. Fairbank supports the view that the lesion is a fracture and bases this conclusion on the following facts: (1) It occurs most frequently in adolescents and young adults indulging in vigorous pastimes. (2) Typical lesions are seen in radiograms and revealed by operation as definite and sometimes recent trauma. (3) A lesion at the typical sites may involve the cartilage only, the detached fragment consisting of normal articular cartilage. In such cases there is a definite history of trauma. (4) There is an entire absence of inflammatory changes in and about the lesion. (5) The gross appearance when operation is performed early suggests nothing but a simple recent fracture. When sufficient time has elapsed for changes to occur, the only ones that do take place are those which would be expected from an effort on the part of the tissues to repair the damage. Exactly similar changes are occasionally found on the more exposed parts of the femoral articular surface where the traumatic origin of the lesions is never disputed.[1]

The following case report is unusual in so far as the lesion involved the entire articular surface of the lower end of the femur, and also in so far as the opposite knee-joint revealed a flattening of the distal femoral articulating surface. This flattening may have resulted from a similar disturbance to the one which we are here describing, but eventually healed without the formation of loose bodies.

CASE REPORT

J. S., female, aged 19, a typist by occupation, was first observed on September 18, 1935, when she complained of a painful left knee. The pain had not been intense at any time nor did it follow any injury or infection. The discomfort had been present on and off, for about one year, but the patient never was disabled at any one time. There was no significant antecedent history of any consequence bearing on this particular disturbance, nor did the patient have any complaints referable to other joints.

Local Examination. The angle of greatest extension was 170 degrees and that of greatest

flexion 35 degrees. The circumference of the left thigh was one inch less than that of the right, and with the left knee one-half inch less

Fig. 1. *X*-ray of anteroposterior view of affected joint.

Fig. 2. *X*-ray of anteroposterior view of normal joint (opposite knee).

than the right. There was no evidence of locking either on examination or in the history. All other joints were normal in every respect.

X-ray examination, on September 26, 1935, disclosed flattening of the external condyle of the right femur with degenerative changes, probably nutritional in type, immediately adjacent to the articular surface. The normal intercondylar notch was not so prominent as one would normally expect. The spinous processes of the tibia were smaller than one observes at this age period.

The anteroposterior view of the left knee revealed marked flattening of both condyles of the femur, a very shallow intercondylar notch, and flattened spinous processes. The external condyle of the femur exhibited a dense shadow about the size of a five-cent piece, fairly regular in outline though somewhat ovoid in shape. On the articular surface of the internal condyle were two moderately dense shadows, surrounded by an area of atrophy. The remainder of the bone was normal in texture. In the lateral view there were three distinct shadows indicative of an osteochondritis dissecans. *X*-rays of the hips showed no evidence of pathology.

The patient was admitted to the New York Hospital on October 7, 1935.

Operation. With the patient anesthetized, a tourniquet was applied about the upper third of the left thigh and the limb prepared in the usual manner. A long Fisher incision was made on the inner aspect of the left knee and the entire joint exposed to view. Exploration revealed a large congested fat pad with thickening of several villi. The articular surface of the femur was brought into complete view by laterally dislocating the patella, and observation disclosed a marked flattening of both femoral condyles. The cartilage covering the articular ends of the femur was loose through-

out although it was attached to the lateral margins of the bone. Through the cartilage there appeared three opaque shadows and these

Fig. 3. *X*-ray of lateral view of affected joint.

opacities were found to be rather dense. These areas made themselves evident through the cartilage, simulating callus formation on the sole of the foot.

Due to the marked loosening of the cartilage of the internal condyle from the underlying bone it was deemed advisable to remove this area. Several holes were drilled through the lower end of the femur to vascularize the region and the raw surface of the femur was gently curetted. The cartilage covering the external condyle of the femur was not quite so loose, and because of this a series of drill holes was made through the cartilage and the adjacent femoral condyle. This segment was allowed to remain intact. The semilunar cartilages were normal in shape and appearance and were not disturbed. The articular surface of the tibia was macroscopically normal, and the crucial ligaments were intact and undisturbed. The wound was closed in layers with silk, and the leg was encased in plaster of paris bandages from the toes to the groin.

The patient remained in the hospital until October 16, and on October 23, the plaster and sutures were removed. The wound had healed by primary intention. Physiotherapy was instituted, an elastic knee corset was applied, and the patient returned to work three months after the operation. She has since been able to get about without discomfort and without a limp, although the atrophy of the thigh and calf has persisted in proportions similar to those noted prior to operation. The knee-joint can now be moved through its normal range, there are no points of tenderness, and at no time has synovitis been present.

Following the operation the specimens were referred to the laboratory for section and the following report was received:

"The specimen is a piece of cartilage measuring 4 by 2.5 by 9.5 cm. It feels bony for about half of its length. The remainder is elastic and pliable. Calcification is so complete that it cannot be cut with a knife. Even the flexible portion is calcified.

"Sections show masses of apparently normal bone in which the marrow is completely replaced by fatty tissue. The bone is covered by a layer of partly calcified connective tissue upon which are superimposed festoons of calcified collagenous tissue which forms a structure not unlike that of the callus seen in fractures. Other sections show cartilaginous tissue exhibiting extensive calcification and a peculiar worm-eaten or scalloped appearance at its margin, enclosing a sort of marrow cavity with imperfectly formed bony spicules at the center. At the margin of this mass of cartilage, papillary projections are found in which cartilage cells lie grouped in cords and masses, and through which course wavy bands of what appears to be compacted fibrin. Some of these papillary projections are surrounded by margins of this material. One bit is apparently completely detached and is made up of three masses of poorly formed cartilage with very irregularly distributed cartilage cells. It is surrounded by a many-layered membrane composed of degenerated synovial cells which present very bizarre shapes and have elongated processes. Here again, the cartilage cells are grouped in almost epithelial fashion in small knots of six or seven up to thirty or more. The cartilage has under-

gone extensive calcification without, however, any evidence of true ossification. Many of the cartilage cells appear to be elongated and drawn out as though they were caught in the process of migration. This picture seems to be compatible with a diagnosis of osteochondritis dissecans.

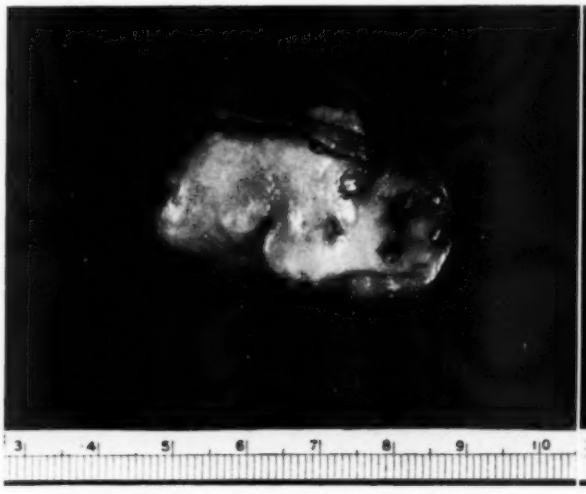

Fig. 4. Specimen, showing articular surface.

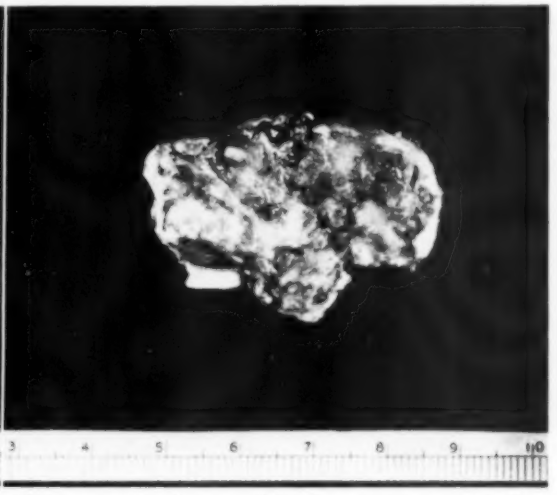

Fig. 5. Specimen, showing non-articulating surface.

"Hemotoxylin-eosin specimens give very little indication of all the interesting changes described above. These were noted in sections stained by Masson trichrome licht-grün."

X-rays of the left knee in the anteroposterior view, taken November 30, 1935, disclose nothing unusual other than the absence of the calcified fragments of bone that were previously present in the neighborhood of the internal condyle of the femur.

X-rays taken July 21, 1936 disclose moderate filling in of the atrophied area of the internal condyle of the left femur. The dissecting ostoechondritis of the external condyle of the femur is still in evidence, particularly in the anteroposterior view, but is not quite so dense in the lateral view as it was on previous occasions.

REMARKS

We are led to believe that this lesion was one of osteochondritis dissecans, simulating osteochondritis of the type of Perthe's disease, or fragmentation of the metatarsal head. The pathology encountered also simulates this latter condition. The condyle of the opposite knee presumably represents a healed lesion.

Osteochondritis dissecans may be looked upon as resembling in great part osteochrondritis of the first decade of life, in which, as a result of faulty nutrition and permanent damage to the underlying vascular structures, a loose segment of bone is thrown off to produce a resemblance to the subchondral fracture previously described. In other words, osteochondritis dissecans may be a late stage of certain forms of osteochondritis juvenilis.

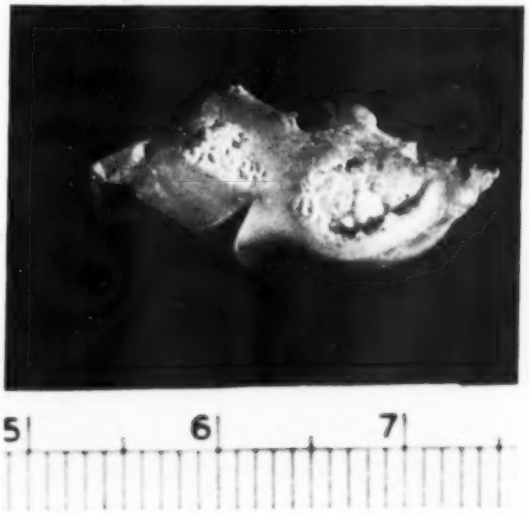

Fig. 6. Cross-section of gross specimen, showing bone nuclei imbedded in cartilage.

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MAL-UNION IN SUPRACONDYLAR FRACTURE OF HUMERUS—RESULT FIFTEEN YEARS AFTER OPERATION

G. T. Tyler, Jr., M.D.

GREENVILLE, SOUTH CAROLINA

IN the December, 1929, issue of this Journal, I reported, with illustrations, the seven year result of operation for the relief of deformity caused by mal-union of a supracondylar fracture of the humerus. The patient is now twenty three years old. He has been examined at intervals since 1928. He has complete extension; and the limit of flexion is a fraction greater than at the last report. A casual observation would discover no defect. On Dec. 9, 1936, another lateral *x*-ray of the elbow was taken in flexion and in extension.

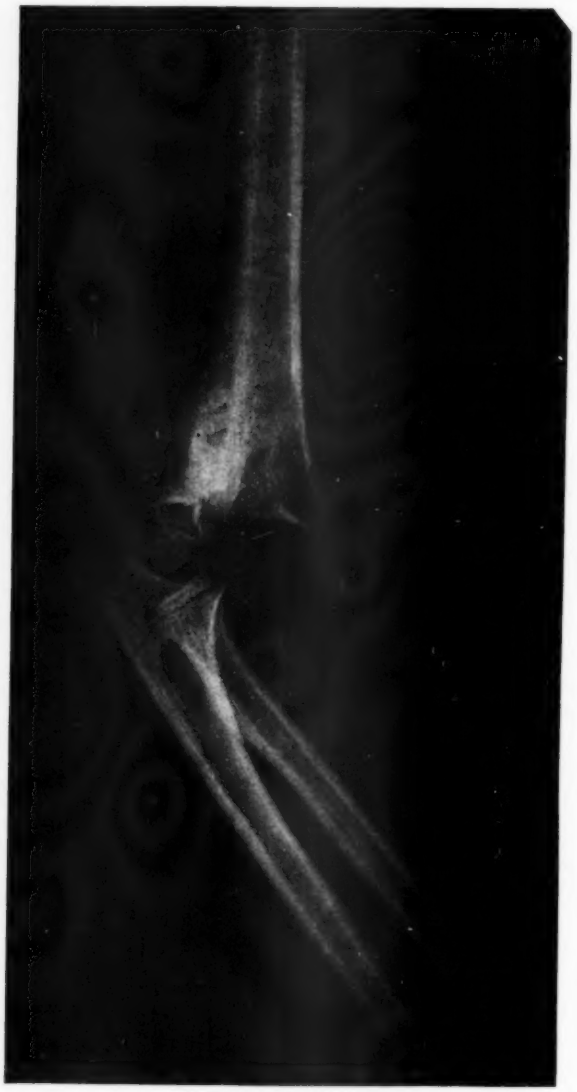


Fig. 1. Taken January 1922.

Study of the *x*-ray films, and comparison with those of 1928, reveal the following changes: in addition to the increased size

Fig. 2.

Fig. 3.

Figs. 2 and 3. Taken January 1922.

Fig. 4.

Fig. 5.

Figs. 4 and 5. Taken November 1928.

and density of the bones, the lower half of the humeral shaft is nearer a straight line, and there is a definite depression at the site of the coronoid fossa. Except for the absence of the normal curve in this end of the bone, there is hardly any deformity. In this case the outcome, both anatomically and functionally, is excellent.

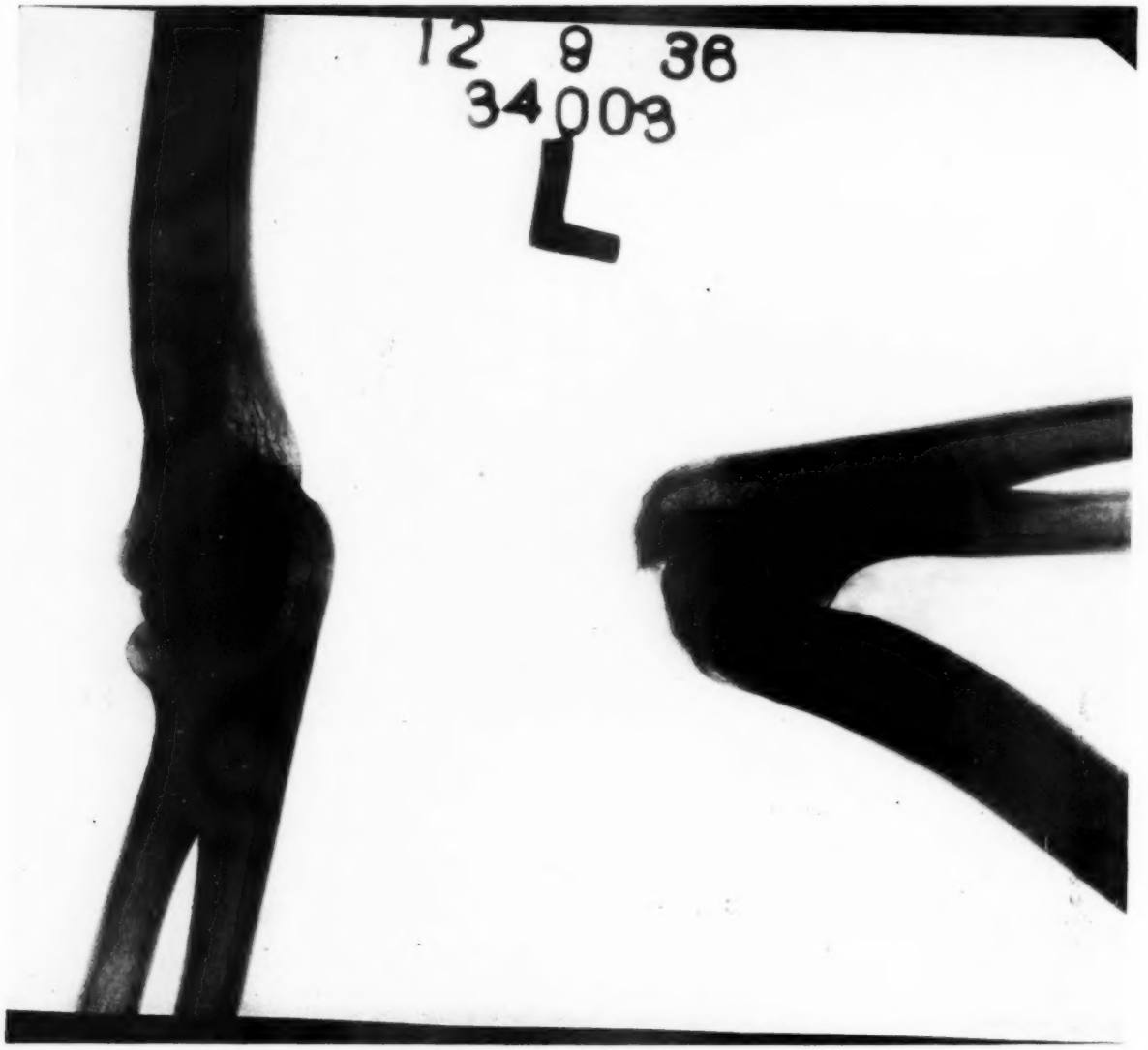


Fig. 6. Taken December 1936.

If the surgeon is willing to wait, Nature will restore the function after minimal operative effort.

NEW INSTRUMENTS

A BRIDGE FOR SUPPORTING THE SCROTUM FOLLOWING HERNIA OPERATION

WALTER C. GRAHAM, M.D. AND BRADLEY L. COLEY, M.D.

NEW YORK CITY

SURGEONS with considerable experience in the operative treatment of hernia and hydrocele are quite familiar with the complication of swelling of the scrotum and its contents which often occurs after these operations. This swelling may be caused by a venous obstruction due to trauma in dealing with the spermatic cord or to extravasation of blood in the scrotal tissues. Whatever the cause may be, support of the scrotum in an elevated position as indicated will limit the swelling and diminish the period necessary for restoration to normal.

FIG. 1. Convenient type of scrotal support for use after hernia, varicocele, or hydrocele operations.

For a number of years the "Bellevue Bridge" of adhesive plaster has been used for this purpose at the Hospita for Ruptured and Crippled, but we find that it has two drawbacks, *viz:* (1) being strapped to the patient's thighs, it tends to limit motion which is desirable as a prophylactic measure against thrombi and phlebitis; and (2) it is difficult to keep the scrotum in its proper place on top of the "Bellevue Bridge" since any flexion of the thighs tends to permit the scrotum to slip down below the bridge.

FIG. 2. Scrotal support in operation on a hernia patient.

About six months ago we devised a bridge to overcome these disadvantages. It is made from a piece of 12-gauge pliable wire about 32 inches in length. The wire is bent in a "V," the ends shaped in a triangle each side of which is about an inch in length (see illustration). An adhesive plas-

ter lap is then placed on the lower 4 inches of the arch and its free edge is rounded so as to fit the perineum. A pliable wire is used so that the bridge may be fitted to each individual patient. The triangles on the ends of the shafts keep the bridge from rotating or slipping. The platform is then padded with gauze and slipped under the scrotum. The wire shafts and triangles are strapped firmly to the dressing with adhesive.

We have tested this bridge for a period of six months at the Hospital for Ruptured and Crippled and have found it entirely satisfactory, as well as convenient and inexpensive.

The illustrations show the mechanism of this apparatus and its use.

MERE reduction of the strangulated hernia by spinal anesthesia is to be condemned unless operation is immediately performed. . . . Even though it is strongly felt that omentum is the sole content of the sac, this should be no deterrent to immediate operation. . . . Obturator and sciatic hernias causing intestinal obstruction are best recognized and reduced through a laparotomy incision.

From—"The Therapeutic Problem in Bowel Obstructions" by Owen H. Wangensteen (Thomas).

THE IMPROVED ALBEE BONE MILL

FRED H. ALBEE, M.D., F.A.C.S.

NEW YORK CITY

THE Albee bone mill consists of a complete set of instruments—as complete as the carpentry mill—which permits the surgeon, under absolutely sterile conditions, to perform the necessary tasks of bone carpentry and cabinet making, with the precision of a machine shop in commercial carpentry. The outfit consists of:

1. Rotary saws of different diameters.
2. Universally adjustable twin-saws, or the cutting caliper.
3. Various types of reciprocal (oscillating) finger-type saws for deep work, scroll work or cutting of segments of any size of circle.
4. End mill cutters for enlarging gutters, mortices, etc., and for starting drill holes on oblique, hard or slippery bone surfaces.
5. Drills of various sizes and types, up to one-half inch neck of the femur drills.
6. Lathe for manufacture of living bone graft pegs and screws of different sizes.
7. Corresponding size drills for pegs or screws.
8. Different size taps, to cut threads on the inside of the drill holes.
9. Die cutters, to make threads upon bone graft pegs of different sizes.
10. Motor-driven oscillating chisels and files.

With the aid of this apparatus, it is possible to make bone graft inlays for treatment of un-united fractures, bone tuberculosis, etc., as well as autogenous bone pegs and screws for various types of reconstruction surgery, chronic fractures, bone plastic surgery, etc. In fact, all types of bone surgery involving the cutting or shaping of bone are rendered possible with this apparatus. It is the only motor-driven outfit which possesses the versatility of circular saws, drills, peg-making devices, screw-making devices and reciprocating cutting tools.

THE BONE PEG AND SCREW THREADER

The bone peg and screw threader has been newly designed to be used in conjunction with the Albee bone mill, in order to turn out bone graft pegs and bone graft screws, which will automatically and accurately fit into drill holes (threaded or not) made by the various sized drills. Both types of bone grafts are made under completely sterile conditions.

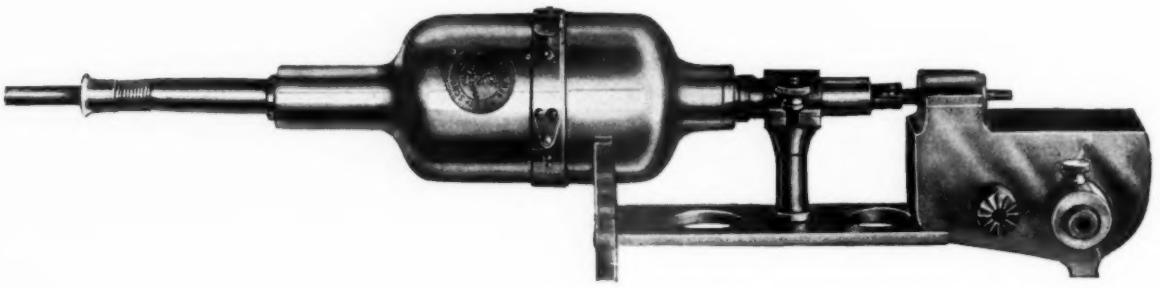
A universal motor (Fig. 1) is attached to a lathe, equipped with cutting tools which are able to form pegs or screws of any desirable size (Fig. 1B and C.) Peg shapers and dies, which may be used interchangeably, form the bone grafts themselves. (Fig. 2D and E.) A concave peg-shaper is first used to shape the graft in conical form (like the action of a rotary pencil sharpener) in order that the graft may be engaged in the peg-shaper and then in the die.

Various sized drills are included, some for pegs and some for screws. (Fig. 2F.) These are differentiated because the relation in size of the peg-shaper to its corresponding drill is different from that of the die to its corresponding drill. The drills are therefore marked "for peg" or "for screw."

After the use of the drills to make appropriate sized holes, taps (Fig. 2G) are used to form threads in the drill holes. These taps, of different sizes to conform to the drills, must be driven slowly for best success, and are accordingly best guided by hand.

The motor is attached to drive the lathe, which consists of a housing with gears, so designed that the peg-shaper revolves at a desirable speed and the die at a much

slower speed, a difference essential for good results. The surgeon gently pushes the bone graft toward the die, forming screw threads upon it to any desired length, while the completed portion protrudes through the far side of the lathe. It is not necessary to push the whole of the graft through the die, since it is usually desirable to leave a head for the screw. To remove the screw from the mechanism, the surgeon merely pulls gently upon the instrument which holds the graft, reversing the rotation of the die and automatically ejecting the screw. This acts in precisely the same way as the reverse gear of an engine transmission, changing the direction of movement of an automobile while the engine is running.



A B C

FIG. 1. A, universal motor attached to lathe, which is able to form, with its cutting tools, pegs and screws of various sizes. B, peg-shaper. C, screw-shaper.

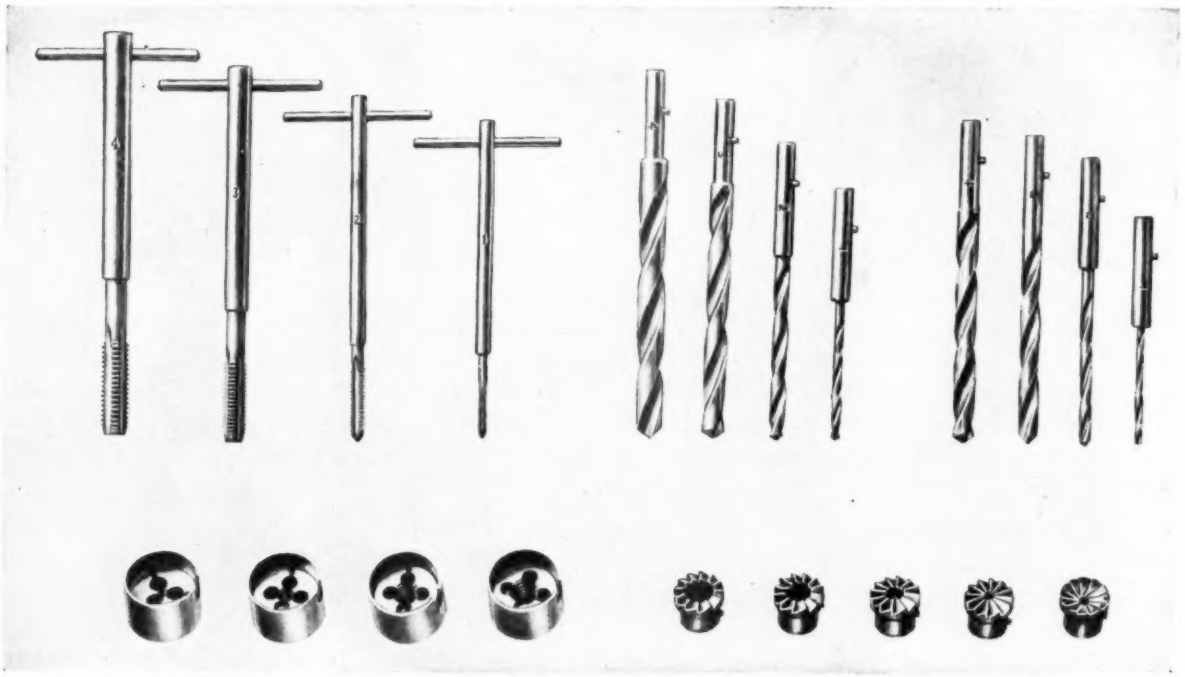


FIG. 2. D, peg-shapers, and E, dies, which may be interchanged to make pegs and screws of any desired size when inserted in the lathe shown in Figure 1. At the extreme right of D is the conical peg-shaper used in first shaping the graft, in order to point it properly. F, drills of various sizes, used in preparing the hole to receive the peg or screw. The drills are differentiated according to whether they are to be used for pegs or screws. G, taps of different sizes for making threads in drill holes. These are most advantageously used by hand, rather than driven by motor.

The apparatus is capable of being sterilized by boiling. It requires little care, although the surgeon should familiarize himself with its parts and capabilities. A screw-plug in the housing enables the lubricant to be changed.

OSCILLATING SAW ATTACHMENT

For special purposes, we have designed an oscillating saw attachment, which has proved of distinct value, particularly in circular osteotomies or other circular bone incisions, and in cutting deeply situated bones.

If there is an adequate marrow cavity on the inside of the bone cortex to cut, one might safely use the oscillating saw. However, most frequently this is not the case; even large marrow cavities may be completely filled with extensive plugs of eburnated bone, as in un-united fractures, etc.

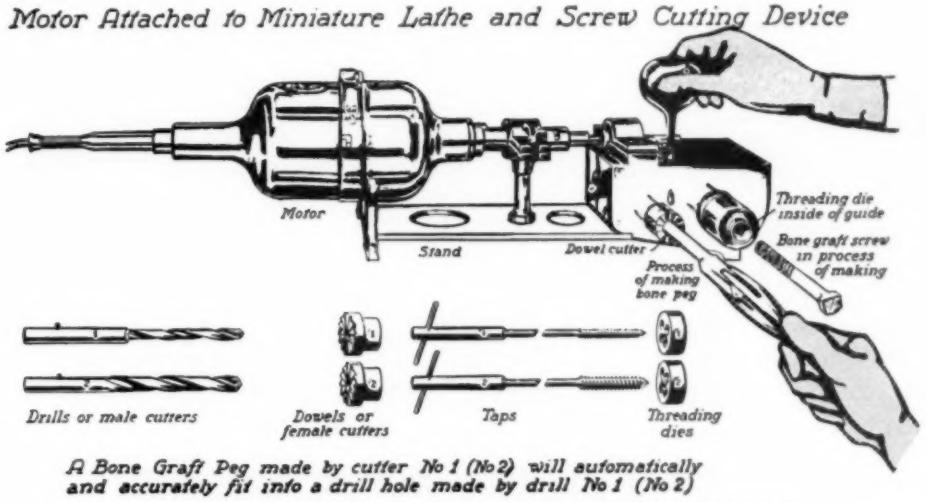

Motor Attached to Miniature Lathe and Screw Cutting Device
Motor
Stand
Dowel cutter
Threading die inside of guide
Bone graft screw in process of making
Process of making bone peg
Drills or male cutters
Dowels or female cutters
Taps
Threading dies
A Bone Graft Peg made by cutter No 1 (No 2) will automatically and accurately fit into a drill hole made by drill No 1 (No 2)

Fig. 3. Method of using the lathe and screw-cutting apparatus.

Fig. 4. The Albee bone mill.

The oscillating type of tool is an adjunct for very special purposes, and we have designed a complete set of these cutting tools, driven directly from the armature shaft, which we find definitely preferable to the flexible shaft. The flexible shaft has been abandoned as a method of power transmission on dental engines, because it causes vibration and chattering of the

cutting tool. This is of particular disadvantage in starting a drill or saw and is extremely undesirable in any precision work, such as straight or mosaic inlays. On the other hand, it is desirable to have the cutting tool attached to the motor armature shaft, as it makes for accuracy and expedites the cutting of cortical bone, which is very hard. A so-called "flexible shaft" which will drive without buckling when a large tool, such as a twin-saw, is driven rapidly through adult bone cortex, must be so large in diameter that it is no longer flexible. Any really flexible shaft will buckle. One instrument has attempted to prevent "dancing of the drill" driven by a flexible shaft by a telescoping gauge which is clumsy in operation.

We have found that the most advantageous power-driven cutting tool for hard surfaces is the rotary one; this is true in bone surgery as well as in manufacturing industries. As a matter of fact, the rotary saw is of particular merit in surgery, as the bone to be cut is surrounded with important soft tissues which should not be traumatized. The rotary saw allows the surgeon to penetrate from one side, as far as he elects, without permitting the cutting tool to penetrate the full thickness of the bone. Thus it cannot injure important soft structures on the other side of the bone, the danger always present in the use of the oscillating finger-type saw.

I should like to thank the Kny-Scheerer Corporation, manufacturers of the Albee bone mill, for their efficiency and painstaking effort in helping to solve the problems in its design and production.

GENERALLY speaking, carcinoma coli is a comparatively slowly growing neoplasm, and if extirpated thoroughly at a reasonably early period, a cure can be hopefully anticipated.
From—"A Short Practice of Surgery" by Hamilton Bailey and R. J. McNeill Love (Lewis).

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

MEDICINE IN FRANCE IN 1837

HENRY M. FEINBLATT, M.D.

Clinical Professor of Medicine, Long Island College Hospital; Attending Physician, and Director of Metabolic Clinic, Kings County Hospital; Pathologist, St. Peter's Hospital

BROOKLYN, NEW YORK

IN the year 1837, Louis Philippe, under the thumb of Metternich, ruled France. This was an interval of great unrest and fundamental political and economic change.

During this period, the medical minds of France were open to new thoughts and readily made new inferences after the presentation of new facts. They had a keen regard for fact rather than authority and leading positions in the medical universities were filled by young men. It was at this time that the system of experimental observation was inaugurated.

The following descriptions, taken from the medical journals published in 1837, would serve to give us an insight as to the character or the medical concepts of the day.

Lalesque described Pellagra as a disease almost unknown to France but observed in Bordeaux and in Italy along the Adriatic Sea.

To quote Lalesque; "In the regions where Pellagra flourishes, the people live in poverty. The dietary includes nothing more than poorly fermented bread, poorly cooked corn, rancid lard and now and then some boiled ham.

"The disease is chronic and may last eight, ten, or fifteen years. It results in marked digestive disturbances with diarrhea, asthenia and typical discoloration of the skin of the hands and feet.

"The skin becomes red, at times uniform in color and peels like a psoriasis. Pellagra is often associated with bronchial disturbances, vaginal discharge and salivation.

"I am much impressed with the relationship of dietary in the production and cure of Pellagra."

MICROSCOPY AND MICROBIOLOGY

The actual subject of microbiology, while conceived, was not yet born. Cagnard and Latour, at this time, showed the proliferation of the small cellular organisms in the fermentation of wine and beer.

Cagnard showed that there was no fermentation after boiling and so attributed the fermentation as due to living particles.

Delpêche and Cosé made observations on the embryo chick and revealed that the circulating fluid, at the earliest stage, was free of blood cells and that the blood cells formed shortly after. Their observations were both microscopic and macroscopic.

It was not until thirty years later that the brilliant Pasteur originated and developed the present day concept of microorganisms and laid the foundation for the subject of bacteriology.

GRIPPE

Recamier, the internist, described a severe epidemic of grippe which presented itself in France in 1837.

"At the central bureau during a period of twenty days, of 1200 hospitalized sick, 1050 were classified as Grippe.

"The disease spreads rapidly and the character of spread is analogous to the epidemic skin eruptions.

"In general, fatigue is very marked, fever, congestion of the eyes and sweating are common; headaches severe, cough painful, respiration rapid. Migratory joint pains and pain in one or both ears are not uncommon symptoms.

Grippe occurred most commonly in the strong, healthy individual and was associated with severe headaches and chest pain. Pulse is slow and resistant. Pleurisy and pneumonia are especially common in the group.

"The second type presents nervous symptoms, insomnia, agitation and asthenia. Pulse is slow and strong. In some cases there is bleeding of the nose and extreme prostration."

SMALLPOX

In February 1837, the various departments throughout France reported on the incidence of smallpox and the results obtained from the use of vaccine obtained from the smallpox pustules. The report was rather incomplete.

The most impressive case was that of Mlle. Fleury, a milkmaid, who on her right hand had three pustules which she contracted from the tits of the cow.

Doctor Bousquet successfully inoculated the contents of these pustules into nine children. He inoculated the old vaccine into other children and gave a comparative report before the commission. In the children he inoculated with the new vaccine, the disease was the same but took a few days longer to develop and the pustules were smaller.

He inoculated both the old and the new vaccine in two children and found the new vaccine to be definitely superior to the old.

In this report, there was record of another young French doctor, Guigon, who successfully immunized six hundred people during a severe epidemic of smallpox by inoculating vaccine obtained from the pustules on the udders of the cow.

Doctor Guigon was criticized for using the cowpox virus without first getting the permission of the commission. He defended himself on the ground that the epidemic would wait for no such permission.

EMPYEMA

It is proper that we attribute the greatest advance in lung physiology to Lavoisier who, in 1777, clearly demonstrated the origin of animal heat and gave to the physiologist a clear conception of oxidation and its relation to respiration and laid the groundwork of general metabolism.

In 1837, when Doctor Cruveilhier gave his treatise on empyema, much confusion as to the treatment of effusion existed and the general concept in vogue was that the influx of air into the pleural cavity would destroy the lung.

By his many experiments on dogs, he found that if the pleural cavities were widely opened on one side, the lung, instead of being compressed, filled and forced its way through the wound in the chest.

While it was generally believed that the animal would die, no matter how wide the openings were made, the animal survived.

He was successful in operating on numerous cases of chronic empyema. In one case, there was recurrent accumulation of fluid after effusion. The patient died after the second operation. An autopsy revealed cancer of the pleura.

The doctor summarized his results as follows:

1. Exposure of the pleural cavity to the air does not cause compression of the lung or inflammation of the normal pleura.

2. The air that does enter is absorbable and stops entering the wound after the animal rests.

3. The wound is readily obstructed by the accumulation of blood; and edema of the soft tissue occurs after exposure.

4. A large opening and complete evacuation is preferable to small openings at repeated cuttings.

5. The accidents that follow operation are due more to the medication employed than to the operation itself.

He here suggested that medicine not be introduced into the pleural cavity in any course of treatment.

DIABETES

In 1674, Willis first called attention to the presence of sugar in the urine in diabetes and the persistence of undernutrition despite an overnutritious diet.

Cullen, the great English pathologist, emphasized the non-sugar type of diabetes or the so-called Diabetes Insipidus.

It was this year that Ambrosiani brought out the relationship between sugar in the blood and sugar in the urine.

Malphighi found that if the renal vessels were tied, an artificial diabetes, with no sugar in the urine, resulted.

Autopsies gave contradictory findings as to the cause of diabetes and the clinicians were not in accord as to the locality of the pathology.

GOITRE

Professor Roux presented the following case history on the extirpation of goitre.

Gerard Jourvain, a youth of 22 with good physical and mental development and good habits observed an enlarged thyroid at 16 years of age. The goitre rapidly enlarged and on local examination was mobile, irregular, bosselated and made up of two distinct lobes connected by the median isthmus.

Pulse was 72, temperature normal. Digestion was good. There was no skin eruption, sore ulceration of throat, epilepsy, headaches, nausea or diarrhea. Patient experienced no difficulty in swallowing.

On May 26th, patient consented to operation.

A longitudinal incision was made in the median line. Skin and muscles were dissected and reflected over the sides of the tumor.

The veins were tied before cutting as many of them were very large, especially at the superior and inferior poles of the tumor. The large arteries, which were attached to the mass, also had to be tied and cut.

In all, forty or forty-seven ligatures were placed. There was considerable bleeding and the loss of blood was estimated at about one and a half liters.

The operation took an hour and ten minutes and the mass was removed in two sections, the first as large as a fist, the second smaller. Several nodules, the size of walnuts were circumscribed at the upper and lower portions of the larger mass. A gauze window was placed over the wound.

The patient withstood the operation very well and readily answered all questions throughout. At no time did he lose consciousness though at several crises, he became faint and turned pale. In these instances water was poured over his face, he was given a little wine, which he promptly vomited.

On the 27th he complained of headaches, insomnia, thirst, colic, constipation, vomiting and slight delirium. On the 28th he complained of sweating, rapid pulse. He was hungry and had no difficulty in swallowing.

During the afternoon of the same day, he became pale and agitated, respiration rapid. Pulse was vibrating at 180. He was bled six ounces and pulse dropped to 140. At 6:00 he died, no convulsion.

Careful and detailed autopsy revealed a local pneumonic patch in the left lung. Death, however, was attributed to the gravity of the operation and not the pneumonia which was only a secondary condition.

A definite relationship existed between disease of the thyroid and lymphoid tissue changes. No definite relationship could be demonstrated between lesion of the thyroid and tuberculosis of the lymphoid tissue, either in this case or in the cases presented by Dupuytren or Graefe.

Despite the opinions of many surgeons of the time, Doctor Roux stated that cancer degeneration of the goitre was very rare.

Extirpation of the goitre was not frequently practiced. Graefe reported three cases; the English doctor, Gouch, reported two; Desault and Dupuytren each reported one.

In four instances in which only one lobe was extirpated, there was recovery. When complete extirpation of the goitre was attempted, death was the result. This report emphasized the gravity of complete extirpation of the thyroid and the lesser danger of single lobe removal.

The danger of injury to the recurrent laryngial, with resulting paralysis, had been emphasized. Such complications were not reported in any of these cases.

When one considers that in the majority of cases, these tumors incur very little inconvenience to health, and that the incidence of malignant degeneration is extremely rare, and that the deformity in the great majority of cases is not serious, it is well to hold back on surgical removals.

To quote Gouch, "I have been asked for my opinion in a number of cases with tumors of this nature, and I cannot recollect one instance in which the tumor, no matter how large, endangered life."

HYPODERMIC

Lafarge, through a series of reports to the Academy of Medicine, described his experiments upon himself and his friends.

By means of a lancet, he introduced various substances under the skin. The substance studied most carefully was the morphine sulphate crystal.

At this time he perfected the hypodermic needle and syringe for the use of subcutaneous medication.

ELECTRO-DYNAMICS

This period marks the publication of André Ampere's monograph on electro-dynamics and also marks the time of his death.

ORTHOPEDICS

Guerin, at this time, presented a new method for treating club feet. He corrected the deformity by first setting the foot in the opposite direction from its original state and then covering it with greased gauze.

This he covered with a layer of plaster and held it in position until the plaster hardened. He then put three or four additional layers of plaster about the foot and leg. These plaster dressings were removed and replaced every eight days.

In his report, he introduced the cases of two children. In both cases, there was double varus equinus. The right foot of each was extremely deformed, the plantar surface perpendicular to the horizon and the dorsal surface completely turned about.

After seven applications of plaster, the feet of both children were in normal position and six days later when the children were brought before the commission, there was no deformity and both children were perfectly capable.

OBSTETRICS

Baudelocque, on April 19, 1837, reported a case of acute hemorrhage of the uterus controlled over an hour by intermittent compression of the abdominal aorta.

CONCLUSION

A historical review is of importance because it shows us how powerful intellects accustom themselves to make new arrangements of thought and break up old ones.

Do we as medical practitioners allow our theories to control our observations? Are we apt to let our opinions mold our understanding?

BOOK REVIEWS

Surgical Pathology of the Diseases of the Neck. By Arthur E. Hertzler, M.D. New York, 1937. J. B. Lippincott Co.

Although this book came to the reviewer with "Compliments of the Author" written on the fly-leaf, and although we have used parts of it for "fillers" in this Journal, we feel the best way we can repay Dr. Hertzler for his kindness is to tell all who read these lines that here is a book that stands at the head of its class. It is well written, the illustrations have been selected with care and are well reproduced, and when one finishes reading the text he feels he has spent some hours in a profitable manner.

This book is very largely a clinical and operating room study. The author in his preface says, "The present tendency to treat all lesions of the neck by irradiation without a definite pathologic diagnosis is to be deplored. What surgeons have learned is in grave danger of being lost by this practice." We concur in these statements.

Dr. Hertzler covers the following field: Preview of the Surgical Affections of the Neck, Hodgkin's Granuloma, Lymphosarcoma, Lympho-Epithelioma, Endotheliomas of the Lymph Glands, Rare Primary Tumors of the Neck, Diseases of the Vestigial Rests, Benign Tumors of the Neck, Diseases of the Salivary Glands, Secondary Tumors of the Neck, and Inflammatory Affections of the Neck.

At the end of each chapter are listed a few books for reference. There is a complete index.

A splendid book for the practicing surgeon and medical student.

The Collapse Therapy of Pulmonary Tuberculosis. By John Alexander, B.S., M.A., M.D., F.A.C.S. Springfield, 1937. Charles C. Thomas. Price $15.00.

There is no doubt but that the large majority of men doing pulmonary surgery will read and reread this book, written by Dr. Alexander, with contributions by Dr. Max Pinner (two chapters on the physiologic principles and the pathology of collapse), Dr. John Blair Barnwell (two chapters on pneumothorax), and Dr. Kirby Smith Howlett (a chapter on oleothorax). We are informed that a considerable number of the details of collapse therapy, as practiced at the University of Michigan Hospital, have been suggested by the author's colleague, Dr. Cameron Haight, and by their present and former associates who have operated upon many of the patients whose cases constitute an important foundation for this book.

It is impossible to go into details and offer a critical review, due to limitation of space. All we can say is that exclusive of a few controversial points, the work as a whole is thorough, encyclopedic in its scope, and at this writing offers the last word on the subject.

It is a beautifully printed and bound book of 705 pages, with 367 illustrations, 1342 bibliographic references, and an ample index.

Arteriovenous Aneurysm. Abnormal Communications between the Arterial and Venous Circulations. By Emile Holman, A.B., B.A.OXON., M.D. New York, 1937. The Macmillan Company. Price $5.00.

This worthwhile volume, one of the series of Macmillan Surgical Monographs under the editorship of Dr. Elliott C. Cutler, which was awarded the 1930 quinquennial Samuel D. Gross Prize by the Philadelphia Academy of Surgery, will appeal to all surgeons and many internists.

The aim of the book is given by the author in his introduction. He tells us that abnormal arteriovenous communications occur so rarely in the experience of one physician that they are to most clinicians a matter of only academic and fleeting interest. Nevertheless, a direct communication between the arterial and venous systems is a unique lesion that illustrates, as no other lesion can, certain physiologic concepts of fundamental importance in the understanding of both the acquired and congenital disorders of the circulatory system.

To quote Dr. Holman: "Direct communications between the arterial and venous systems may occur as a developmental anomaly or as an acquired lesion due to trauma. In congenital cardiac disease, the persistent ductus arteriosus, the patent interventricular septum, and the foramen ovale provide examples of venoarterial

and arteriovenous communications which have the same physiologic effects upon the circulatory system as the peripheral fistula."

Feeling that this clinical problem, so poorly understood, illustrated the need of experimental laboratory work, and following John Hunter's dictum, "Don't speculate, try it and see," the author found it desirable to present the experimental manifestations of a fistula first, in order that the reader may understand more clearly the need and the character of the treatment to be undertaken clinically for its elimination.

We feel that this book of 244 pages (including 79 illustrations, a bibliography, and an index) is an important work, and should be read by many surgeons and physicians.

Radiation Therapy. Its Use in the Treatment of Benign and Malignant Conditions. By Ira I. Kaplan, B.S., M.D. New York, 1937. Oxford University Press. Price $10.00.

This book was written to give the student, the general practitioner and the specialist an understanding of the fundamentals of irradiation. It enumerates the conditions for which this form of therapy is of value. The book supplies rules and suggestions for the clinical care and handling of patients referred for treatment. The author, in discussing radiation therapy, tells us he is giving the reader the results of his own experience in treating patients in both special cancer and general hospitals as well as in private practice.

While Dr. Kaplan has made no attempt to evaluate or to present all the different methods in use by other therapists, he has drawn freely on such as have come to his attention through contacts with other workers or by reason of his position as Editor of the Yearbook of Radiology.

Anyone interested in radiation therapy will do well to study this work. Its 558 pages abound in solid facts and suggestions. The 198 illustrations are excellent. There is a bibliography at the end of each chapter, and an index at the end of the volume.

In this day and age when every Tom, Dick and Harry, lured by the selling-talks of salesmen, purchases an *x*-ray treatment outfit and hires radium from commercial companies, he should at least own this volume and learn certain parts by heart before attempting to treat benign or malignant growths. Perhaps some of these men will be enabled to attain the results they seek, and obtain a working idea of the technique of radiation therapy.

Dr. Kaplan has done the profession a great good in offering it this book.

The Roentgenologist in Court. By Samuel Wright Donaldson, A.B., M.D., F.A.C.R. Springfield, 1937. Charles C. Thomas. Price $4.00.

The author truly says, "Very few physicians are at all interested in legal matters, and especially not in those with a medical angle . . . Every physician is sooner or later called upon to act as a witness—either as an ordinary witness or in the capacity of an expert. . . . Or, more important to him, he may be a defendant in a malpractice suit or be called as a witness in such a suit brought against one of his colleagues. . . . In view of such facts, part of this book has been devoted to malpractice prophylaxis in its relation to legal procedure. Considerable space has also been given over to the novice witness—for the benefit of those who are called upon to appear in court only occasionally . . . Since many roentgenologists are employed by institutions or groups, or have formed partnerships, the author has deemed it proper to include a discussion of some of the legal problems of these relationships and of the accompanying civil liability."

Dr. Donaldson lives up to his promises. He has made a dry subject alive and interesting. The pages abound in good advice and counsel. Naturally, roentgenologists, unless they are veterans in court work, will do well to read and then reread many times this book. And other physicians will find it profitable to learn the lessons offered.

There is a bibliography, an index, and a table of cases.

AUTHOR INDEX TO VOLUME XXXIX

Albee, Fred H., 657
Anderson, Roger, 538

Balensweig, Irvin, 127, 648
Barrington-Ward, Sir Lancelot, 229
Baskin, Louis J., 151
Beekman, Fenwick, 312
Benson, Clifford D., 267
Berry, Frank B., 12
Best, R. Russell, 533
Bettman, Adalbert G., 156
Biskind, Gerson R., 112
Bowen, Arthur, 4
Boyd, Phillips L., 641
Brown, James Barrett, 452
Byars, Louis T., 452

Campbell, Meredith F., 438
Carraway, B. M., 576
Carraway, C. N., 576
Clark, Howard C., 631
Coley, Bradley L., 334, 655
Collins, J. Lloyd, 607

Davis, Benjamin F., 512
Day, Robert V., 100
Diamond, Louis K., 400
Donaldson, J. K., 37
Donovan, Edward J., 377

Emmert, Frederick V., 581

Falk, Henry C., 185
Fein, M. J., 27
Feinblatt, Henry M., 661
Fennel, E. A., 121
Ferguson, Charles Foss, 429
Findlay, Robert T., 46
Forrester, C. R. G., 552

Gaston, Cecil D., 172
Gilbride, John J., 617
Glasser, S. Thomas, 120
Goldschmidt, Siegfried, 581
Gorrell, R. L., 62
Graham, Walter C., 655
Gross, Robert E., 234

Haggland, P. B., 171
Hamm, Frank Coleman, 77
Hanan, J. T., 27
Hanby, John Estes, 135
Harris, Franklin I., 112
Herzfeld, Gertrude, 422
Heyd, Charles Gordon, 18
Hicken, N. Frederick, 533
Hochberg, Lew A., 561
Horneff, J. Arthur, 23
Howard, Tasker, 175

Ingraham, Franc D., 342

Jones, T. Banford, 382

Kessler, E. E., 100
Knoepp, Louis F., 145
Kross, Isidor, 610
Kutzmann, A. A., 100

Ladd, William E., 227, 234
Lanman, Thomas H., 249
LeMesurier, A. B., 458
Loman, Julius, 479
Lowsley, Oswald S., 168

MacCollum, Donald W., 275
MacGuire, D. Philip, 527
Magrath, Joseph L., 148
Maliniac, Jacques W., 54
Manheim, Sylvan D., 86
Marks, Mark M., 86
Martin, Harry W., 100
Masland, H. C., 163
McCarthy, P. A., 148
Miller, J. K., 34, 67
Moersch, Herman J., 521
Moore, W. D., 172
Morton, John J., 382
Munger, Arbor D., 584

Newman, Howard E., 607

Ober, Frank R., 319
O'Brien, Frederick W., 95
Orr, Louis M., 602

Penberthy, Grover C., 267
Peterson, Andrew P., 168
Peterson, Richard L., 334
Phaneuf, Louis E., 620
Prather, George C., 589

Rachlin, William, 178
Robertson, D. E., 327
Ronchese, F., 635

Sappington, S. W., 23
Sauer, Paul K., 646
Schenck, Samuel George, 561
Schwartz, Joseph, 70
Seidler, V. B., 27
Snell, Albert M., 521
Stern, Elias Lincoln, 495

Teller, Frank, 151
Thorndike, Augustus, Jr., 429
Tyler, G. T., Jr., 652

Vernon, S., 130
Vestal, Paul W., 614

Wagner, Lewis Clark, 135
Waters, Ralph M., 470
Weisengreen, H. H., 133
White, Alfred S., 112
White, Charles S., 607
Wilson, F. L., 62
Wolf, Josef, 42, 557
Wollner, Anthony, 627

Young, Thomas O., 104

SUBJECT INDEX TO VOLUME XXXIX

(Bo.B.) = Bookshelf Browsing; (E.) = Editorial; (S.) = Serial

Abdomen
  adhesions, 37
  construction of supports, 42
  supports for, 42
Abscess, Brodie's, 135
Adhesions
  abdominal, 37
  causes, 37
  cotton sponges and, 37
  gauze sponges and, 37
Alkyl-dimethyl-benzyl ammonium chloride, clinical use, 607
Amputations
  in poor-risk patient, 46
  of lower extremity, in aged, 46
Analgesia, in labor, 581
Anemia, congenital hemolytic, 403
    splenectomy in, 403
Anesthesia
  eucupin in, 86
  in amputations, in aged, 49
  in children, 474
  in proctology, 86
  intravenous, 576
Anomalies
  congenital branchiogenic, 234
  intestinal, in children, 382
Antisepsis, zephiran in, 607
Appendicitis
  conditions diagnosed as, 18
  differential diagnosis, 18
  histologic diagnosis, 23
  histology, 23
  traumatic, 148
Appendix
  plexiform neuroma of, 27
  traumatized, 148
Arterial pressure, cerebrospinal fluid pressure and, 483
Atelectasis, in children, surgical treatment, 249
Atresia, intestinal, 382

Biliary colic, treatment with glyceryl trinitrate, 533
Bleeding, in cesarean section, control of, 62
Blood flow, cerebral, factors affecting, 489
Bone mill, improved, 657
Bone tumors, primary, in children, 334
Book Reviews:
  Arteriovenous Aneurysm. Abnormal Communications between the Arterial and Venous Circulations (Holman), 665
  Collapse Therapy of Pulmonary Tuberculosis (Alexander), 665
  Everyday First Aid (Cobb), 182
  Management of Fractures, Dislocations and Sprains (Key and Conwell), 182
  Post-Graduate Surgery (Maingot, Ed.), 182
  Preventive Medicine and Hygiene (Vol. XII of Practitioner's Library of Medicine and Surgery), 183
  Radiation Therapy. Its Use in the Treatment of Benign and Malignant Conditions (Kaplan), 666
  Roentgenologsit in Court (Donaldson), 666
  Surgical Pathology of Diseases of the Neck (Hertzler), 665
Book Reviews:
  Textbook of the Practice of Medicine (Price, Ed.), 182
Braces, for scoliosis, construction of, 557
Brain, injuries of, physiologic indications in treatment, 512
Branchial clefts, congenital anomalies originating in, 234
Branchial cysts, congenital, 234
Breast
  anatomy, 54
  carcinoma of, lymphatic injection in, 617
    x-ray in, 617
  deformities of, 54
  keloid of, 635
  physiology, 54
  plastic operation on, 54, 614
    anatomy, 54
    physiology, 54
  scleroderma of, 635
  sclerosis, 635
Bridge, for scrotal support, after hernia operation, 655
Brodie's abscess, 135
  diagnosis, 135
  occurrence, 135
  pain distribution, 135
Bronchiectasis, in children, surgical treatment, 249
Bullets, perforating intestines, 646
Burns
  contractures in, 287
  fresh, treatment of, 276
  homogenous Thiersch grafting in, 156
  in children, treatment, 275
  scars in, treatment, 307
  unhealed, treatment, 282

Calculi
  staghorn, "V" type nephrotomy in, 589
  ureteral, management, 584
Carcinoma
  gastric, 527
    gastroscopy in, 524
  of breast, lymphatic injection in, 617
    x-ray in, 617
  of Fallopian tube, 620
Carpal bones, fracture of, splints for, 163
Cerebrospinal fluid pressure
  arterial pressure and, 483
  drugs and, 484
  osmotic forces and, 486
  venous pressure and, 480
Cervix, polyps of (S.), 222
Cesarean section
  "bloodless," 62
  control of bleeding in, 62
  pituitrin in, 62
Children
  anesthesia for, 474
  burns in, treatment, 275
  cervical tuberculous glands in, 229
  chronic pulmonary suppuration in, treatment, 249
  compound fractures in, 312
  congenital intestinal malformations in, 382
  dilated ureter in, 438

Children, empyema in, treatment, 267
fractures and dislocations of elbow in, 327
incarcerated hernia in, 429
indications for splenectomy in, 400
intraspinal tumors in, 342
osteomyelitis in, 319
pain relief in, 470
primary bone tumors in, 334
Cleft palate, operative treatment, 458
Compound fractures, in children, 312
treatment, 312
Congenital anomalies
branchiogenic, 234
intestinal, 382
Congenital hemolytic anemia, 403
Congenital hypertrophic pyloric stenosis, 377
Contraception, dangers of intra-uterine pessaries in, 631
Contractures, from burns, plastic repair, 287
Cotton sponges, adhesions and, 37
Craniovertebral dynamics, 479

Decade ends (E.), 1
Dental explorer, in pinch grafting, 171
Diaphragmatic hernia, 4
Dislocations, of elbow, in children, 327
Dynamics, craniovertebral, 479
Dyspareunia (S.), 221

Editorials:
A Decade Ends, 1
Dangerous Legislation, 477
The Trend of Pediatric Surgery, 227
Elbow, fractures and dislocations, in children, 327
Empyema
acute, non-tuberculous, 561
thoracic, complications, 561
roentgenologic considerations, 561
sequelae, 561
in children, treatment, 267
Endocarditis, bacterial, renal infarcts and, 70
Endocervicitis (S.), 186
Endoscope
Lowsley-Peterson, uses, 168
universal, 168
Eucupin
in proctology, 86
in pruritus ani, 86
Evisceration, as post-operative complication, 610
Exostosis, osteogenic sarcoma from, 121

Fallopian tube, primary carcinoma of, 620
Female, sterility in, pitfalls in diagnosis, 627
Femur
distal end, osteochondritis dissecans of, 648
shaft fractures, ambulatory treatment, 538
fracture table reduction, 538
Foreign bodies, in uterus, 631
Fractures
compound, in children, 312
treatment, 312
of carpal bones, splints for, 163
of elbow, in children, 327
of femoral shaft, ambulatory treatment, 538
of humerus, mal-union in, 652
of mandible, 133
of proximal phalanx of thumb, 130
of tarsal bones, 163
Fractures, table, in reduction of femoral shaft fractures, 538
France, medicine in, in 1837 (Bo.B.), 661

Gall-bladder, glyceryl trinitrate and, 533
Gastritis, gastroscopy in, 521
Gastroscopy
in benign gastric tumors, 524
in diagnosis of gastric disease, 521
in gastric carcinoma, 524
in gastric ulcer, 523
in gastritis, 521
Gaucher's disease, splenectomy in, 416
Gauze sponges, adhesions and, 37
Genitourinary tuberculosis, 67
Glands, cervical, tuberculosis of, 229
Glyceryl trinitrate
in post-operative biliary tract disease, 533
in treatment of biliary colic, 533
Gonorrhea, in female, treatment (S.), 212
Grafting, pinch, dental explorer in, 171
Guthrie, Samuel, the forgotten man (Bo.B.), 178
Gynecology, practical (S.), 185

Hemangioma
interstitial radiation in, 452
treatment, 452
Hemorrhage, gastric, splenomegaly in, 412
Hernia
bridge for post-operative scrotal support, 655
diaphragmatic, 4
in infancy, 422
incarcerated, 429
incarcerated inguinal, in children, 429
injection treatment, solutions used in, 112
Histology, in appendicitis, 23
Hormones, in treatment of prostatism, 100
Humerus, supracondylar fracture, mal-union in, 652
Hyperthyroidism
continuing, 104
recurrent, 104

Infants
hernia in, 422
incarcerated, 429
Infarcts, renal, 70
bacterial endocarditis and, 70
Inhibin, prostatism and, 100
Intestine
atresia of, 382
congenital malformations, 382
multiple bullet perforations, 646
tuberculosis of, 34
Intravenous anesthesia, 576
Isoamylhydrocupreine, in proctology, 86

Keloid, of breast, 635
Kidney, infarcts, 70
bacterial endocarditis and, 70
Knee, pedunculated lipoma of, acute torsion, 127

Labor, analgesia in, 581
Legislation, dangerous (E.), 477
Legs, amputation of, in aged, 46
Lipoma
of knee, acute torsion, 127
pedunculated, of knee, 127

Lowsley-Peterson endoscope, 168
Lungs, chronic suppuration, surgical treatment, 249
Lymphatics, *x*-ray visualization, in breast cancer, 617

Mandible, gunshot fracture, 133
Medicine
  in France, in 1837 (Bo.B.), 661
  100 years ago (Bo.B.), 175
Monolate, in treatment of varicose veins, 120

Neck, tuberculous glands, in children, 229
Nephrotomy, "V" type, for staghorn calculus, 589
Nervous system, diseases of, intraspinal injection of vitamin B_1 in, 495
Neuroma, plexiform, of appendix, 27

Office gynecology (S.), 185
Office urology (S.), 223
Omentum, torsion of, 151
Osteochondritis dissecans, of distal end of femur, 648
Osteomyelitis
  in children, 319
    over two, 320
    treatment, 319
    types, 319
    under two, 319

Pain, in children, relief of, 470
Palate, cleft, operative treatment, 458
Parotid tumors, mixed, 641
Pediatrics, surgery in, trends (E.), 227
Pessaries, intra-uterine, dangers of, 631
Pinch grafting, dental explorer in, 171
Pituitrin, in cesarean section, 62
Plexiform neuroma, of appendix, 27
Polyps, cervical (S.), 222
Practical clinical gynecology (S.), 185
Proctology
  anesthesia in, 86
  eucupin in, 86
Prostate
  hypertrophy, hormone treatment, 100
  transurethral resection, 77
Prostatism, inhibin and, 100
  testosterone in, 100
Prostatitis, chronic, treatment, 602
Pruritus ani, treatment with eucupin, 86
Purpura hemorrhagica, splenectomy in, 414
Pyloric stenosis, congenital hypertrophic, 377
Pylorus, congenital stenosis, 377
Pyopneumothorax, tuberculous, 145

Radiopaque substance, in breast carcinoma, 617
Radiotherapy, supervoltage, 95
Radium, interstitial, in treatment of hemangioma, 452
Resection, prostatic, 77

Sarcoma, osteogenic, from exostosis, 121
Scars, from burns, treatment, 307
Scleroderma, of breast, 635
Sclerosis, of breast, 635
Scoliosis, construction of braces in, 557
Scrotum, bridge for support of, after hernia operation, 655
Sex hormones, in prostatism, 100
Sigmodal, as analgesic in labor, 581
Sitz bath, portable, 172
Skin, Thiersch grafting, 156
Spine
  injection of vitamin B_1 in, 495
  tumors of, in children, 342
Splenectomy
  in children, indications for, 400
  in congenital hemolytic anemia, 403
  in Gaucher's disease, 416
  in purpura hemorrhagica, 414
  technique, 400
Splenomegaly, in gastric hemorrhage, 412
Splint, for carpal bone fractures, 163
Sponges
  adhesions and, 37
  cotton base, 37
Stenosis
  congenital pyloric, 377
  intestinal, 382
Sterility, in female
  pitfalls in diagnosis, 627
  surgical treatment, 627
Stomach
  benign tumors, gastroscopy of, 524
  carcinoma of, 527
    gastroscopy in, 524
  diseases of, gastroscopy in, 521
  ulcer of, gastroscopy in, 523
Stones, ureteral, management, 584
Supports, abdominal, construction of, 42
Suppuration, chronic pulmonary, surgical treatment, 249
Surgery, in pediatrics, trends (E.), 227
Surgical problems, 180
Syphilis, legislation on (E.), 477

Tarsal bones, fractures of, 163
Tendon suture
  delayed, end results, 552
  early, end results, 552
Testosterone, in prostatism, 100
Thiersch grafting, in burns, 156
Thorax
  acute empyema, complications, 561
    sequelae, 561
    roentgenologic considerations, 561
  wounds of, 12
Thumb, proximal phalanx, fracture of, 130
Trauma, in appendicitis, 148
Trichomonas vaginalis vaginitis (S.), 201
Tuberculosis
  genitourinary, 67
  intestinal, 34
  of cervical glands, in children, 229
  pyopneumothorax in, 145
Tumors
  intraspinal, in children, 342
  mixed, of parotid type, 641
  of bone, in children, 334

Ulcer, gastric, gastroscopy in, 523
Ureter
  calculi in, management, 584
  dilatation of, in children, 438
Uterus, foreign bodies in, 631

Vaginitis, senile (S.), 219
Veins, varicose, sclerosing drug for, 120
Venous pressure, cerebrospinal fluid pressure and, 480
Viscera, thoracic, wounds of, 12
Vitamin B_1, intraspinal injection
  in diseases of nervous system, 495
  in intractable pain, 495

Vitamin B_1, in von Recklinghausen's disease, 500
Von Recklinghausen's disease, intraspinal injection of vitamin B_1 in, 500
Vulvovaginitis, in children (S.), 217

Warts, venereal (S.), 220
Wounds, of thoracic viscera, 12

X-ray
in acute thoracic empyema, 561
in breast cancer, 617
supervoltage, 95

Zephiran, clinical use, 607